LANE MEDICAL LIBRARY STANFORD
I1246 .V41 1925 OLD
The medical aspects of chemical warfare
24503132194

LANE

MEDICAL LIBRARY

LEVI COOPER LANE FUND

LANE MEDICAL LIBRARY OF
STANFORD UNIVERSITY
300 PASTEUR
PALO ALTO, CALIFORNIA

Digitized by the Internet Archive
in 2025

https://archive.org/details/medicalaspectsof0000edwa

THE MEDICAL ASPECTS OF CHEMICAL WARFARE

AIRPLANE PHOTOGRAPH OF FIRST GAS ATTACK MADE BY THE GERMANS AT YPRES BETWEEN LANGEMARCK AND BIXSCHOOTE AT ABOUT 5:00 P.M. APRIL 22, 1915

In this attack 180 tons of chlorine were liberated from 6000 containers on a front of 6 kilometers. The attack lasted about five minutes. About 5000 men were killed and there were many times more casualties. Fifty guns were lost, and the position had to be abandoned.

THE MEDICAL ASPECTS OF CHEMICAL WARFARE

BY

EDWARD B. VEDDER

LIEUT. COLONEL, M.C., U. S. A.

WITH A CHAPTER ON THE

NAVAL MEDICAL ASPECTS OF CHEMICAL WARFARE

BY

DUNCAN C. WALTON

LIEUT. COMMANDER, M.C., U. S. N.

BALTIMORE

WILLIAMS & WILKINS COMPANY

1925

COPYRIGHT 1925
WILLIAMS & WILKINS COMPANY

Made in United States of America

Published January, 1925

COMPOSED AND PRINTED AT THE
WAVERLY PRESS
BY THE WILLIAMS & WILKINS COMPANY
BALTIMORE, MD., U. S. A.

LIBRARY

CONTENTS

PREFACE ix
INTRODUCTION xi

CHAPTER I

OUTLINE HISTORY OF CHEMICAL WARFARE 1

CHAPTER II

PHYSICS AS APPLIED TO CHEMICAL WARFARE 11

CHAPTER III

ELEMENTARY CHEMISTRY OF CHEMICAL WARFARE 21

CHAPTER IV

METEOROLOGY 40

CHAPTER V

CHARACTERISTICS OF GAS OR SMOKE CLOUDS 62

CHAPTER VI

CLASSIFICATION OF CHEMICAL WARFARE AGENTS 68

CHAPTER VII

THE PULMONARY IRRITANTS—CHLORINE, PHOSGENE, CHLORPICRIN 77

CHAPTER VIII

THE VESICANTS—MUSTARD, LEWISITE 125

CHAPTER IX

THE LACHRYMATORS—CHLORACETOPHENONE, BROMBENZYLCYANIDE 167

CHAPTER X

IRRITANT SMOKES, DIPHENYLCHLORARSINE, DIPHENYLAMINECHLORARSINE 173

CHAPTER XI

MISCELLANEOUS GASES 184

CHAPTER XII

INDIVIDUAL PROTECTION—THE GAS MASK. PROTECTIVE CLOTHING 196

CHAPTER XIII

COLLECTIVE PROTECTION 216

64816

CHAPTER XIV

Organization of the Medical Department for Dealing with Gas Casualties 233

CHAPTER XV

Ultimate Condition and Disposal of Gassed Cases 244

CHAPTER XVI

The Naval Medical Aspects of Chemical Warfare 264

CHAPTER XVII

The Treatment of Certain Respiratory Diseases by Chlorine 294

Appendix 305

Author Index 317

Subject Index 319

LIST OF ILLUSTRATIONS

Airplane photograph of first gas attack made by the Germans at Ypres between Langemarck and Bixschoote at about 5:00 p.m. April 22, 1915 ..... *frontispiece*
Bullet wound of face (plate 1) xii
Preparing battery of Livens projectors (plate 2) 4
Livens projectors ready to fire (plate 2) 4
Firing battery of Livens projectors (plate 3) 4
Explosion of 14 Livens projectors fired at night (plate 3) 4
Three-inch stokes mortar (plate 4) 4
Trench mortar (plate 5) 10
Alphatic series (chart 1) 38
Aromatic series (chart 2) 39
Lungs of dog dying 12 hours after phosgene gassing. Extreme oedema with patches of acute emphysema (plate 6) 90
Blood pressure chart (fig. 1) 101
Heart and lungs of dog dying 12 hours after chlorpicrin gassing. Extreme oedema of lungs; dilation of heart (plate 7) 110
Specimen from fatal case of mustard poisoning, showing false membrane on tongue and in trachea (plate 8) 136
Similar case showing necrotic mucosa, sloughing and ulceration (plate 9) .. 136
Experimental mustard burn, 24 hours after application (plate 10) 142
Mustard burn, 24 hours after contact (plate 10) 142
Same mustard burn, four days after contact (plate 10) 142
Chloracetophenone smoke candles (plate 11) 170
Smoke cloud produced by candles (plate 12) 174
Airplane producing smoke (plate 13) 176
Apparatus used in testing irritant smokes on man (plate 14) 178
To sling the mask. First position (plate 15) 208
To sling the mask. Second position (plate 15) 208
To sling the mask. Third position (plate 16) 208
Mask drill. Helmet with strap under chin. One (plate 16) 208
Mask drill. One continued (plate 17) 208
Mask drill. Two (plate 17) 208
Mask drill. Two continued (plate 18) 208
Mask drill. Three (plate 18) 208
Mask drill. Five (plate 19) 208
Mask drill. Six (plate 19) 208
Mask drill. Helmet with strap back of head. One (plate 20) 208
Mask drill. Two (plate 20) 208
Mask drill. Three (plate 21) 208
Mask drill. Test for gas (plate 21) 208
Mask drill. Remove mask (plate 22) 208

Mask drill. Remove mask continued (plate 22)..... 208
Mask inspection drill. "Prepare for inspection" (plate 23)..... 210
Inspection mask. One (plate 23)..... 210
Inspection mask. Two (plate 24)..... 210
Inspection mask. Three (plate 24)..... 210
Inspection mask. Four (plate 25)..... 210
Inspection mask. Five (plate 25)..... 210
Inspection mask. Six (plate 26)..... 210
Inspection mask. Seven (plate 26)..... 210
Lesions caused by phosgene (plate 27)..... 262
Lesions caused by chlorpicrin (plate 28)..... 262
Lesions caused by mustard (plate 29)..... 262
Smoke screen by destroyers (plate 30)..... 268
Airplane smoke curtain (plate 31)..... 270
Smoke curtain as demonstrated at New York City Harbor, May 2, 1924 (plate 32)..... 272
Burst of 100 pound phosphorus bomb (plate 33)..... 274
Chlorine Inhalator (plate 34)..... 300

PREFACE

Judging by my own ignorance when I was assigned to the study of the medical aspects of chemical warfare, instruction in this subject is very necessary for medical officers, both on the active list and in the reserve. It was at first hoped that a school for medical officers might be organized at Edgewood Arsenal. This has been found to be impracticable, and in any case only a comparatively small number of officers could be sent to such a school. I have, therefore, attempted to gather together in the shortest possible space, the information that medical officers ought to have on this important subject; hoping that it will be like a compressed tablet, which if not palatable at least goes down easily. In obtaining this information I have been especially indebted to the "Notes on Chemical Warfare" prepared by the Chemical Warfare School at Edgewood Arsenal, and to Volume II of the Official History of the War, London, 1923, which includes a section on the "Medical Aspects of Gas Warfare."

I also wish to acknowledge my obligation to Lieut. Commander Duncan C. Walton, M.C., U. S. N., for suggestions and valuable criticism, and for writing the chapter on the Naval Medical Aspects of Chemical Warfare.

With the exception of several photographs from the Army Medical Museum, Washington, D. C., I am indebted for all photographs to Mr. Carl H. Brown of the photographic laboratory, Edgewood Arsenal.

INTRODUCTION

There has been and there still is considerable prejudice against the use of gas in warfare. The arguments for and against the use of gas are interesting to the medical officer. For although he has naturally no connection with the offensive use of gas, he must be prepared to care for gas casualties should gas be used against our army. For this reason the medical officer is interested in the probability that gas will be used in future warfare, and it will be well to examine the facts as briefly as possible to determine that probability.

The prejudice against the use of gas apparently proceeds chiefly from three sources. Primarily from the fact that the Germans who first used gas thereby violated their treaty obligations, as they had systematically violated all of their other treaty obligations as well as the rules of warfare supposedly observed by all civilized nations. Gas warfare has most naturally but quite illogically shared the obloquy that fell upon the Germans. Secondly to the fact that gas is a new weapon. History helps us to understand this prejudice, for other weapons when first developed met the same opposition. When gunpowder was first introduced, the clumsy bombards that were the ancestors of our modern artillery were described as noisy, stinking engines that spoiled good archery. When the matchlock was used it was seriously claimed that "soldiers were struck down by abominable bullets which had been discharged by cowardly and base knaves, who would never have dared to meet true soldiers face to face and hand to hand." Yet such weapons have remained in the face of prejudice and have undergone a marvellous development. The rifle, the machine gun and other similar weapons remained because they were the most efficient weapons that could be produced. Survival in weapons can be shown to be due consistently to efficiency. When a man or a nation is to be subjected to a combat whose result may well be fatal, the weapons that are deemed the best will be chosen.

Finally, prejudice exists against the use of gas owing to the belief the the use of gas is barbarous and inhumane. Of course gas warfare is barbarous and inhumane, but so is all warfare. Is gas warfare

more barbarous and inhumane than the other forms of warfare? Does it cause more loss of life or more suffering? Are the after effects of gas more serious than the effects of other forms of warfare? The facts indicate that gas warfare is more humane than other forms of warfare. Gas causes a smaller proportion of deaths than other weapons. Only 1.73 per cent of our total gas admissions resulted in death. Yet 8.26 per cent of our gunshot admissions resulted in death. If deaths on the field were considered, the disproportion would be many times greater, since many wounded men die on the field, but few gassed cases.

Gas causes less suffering than wounds. Chlorine, the first gas used, is a powerful irritant and causes strangulation and considerable pain. As it was used against troops who were totally unprotected, the mortality in the first attack was also high. In all probability chlorine will never be used again as a war gas. Its place was taken later in the war by more poisonous gases which were far less painful. A high concentration of phosgene or chlorpicrin causes instant collapse with no suffering. With lower concentrations there is no pain. In fact it is sometimes difficult to make a man gassed by phosgene understand that he must give up and lie down. The great superiority of mustard gas was largely due to the fact that the troops did not know they were being gassed. The average man gassed by mustard reported to the first aid station about eight hours after he had been gassed. From a military standpoint, a gas that poisons without pain is far more effective than a painful gas, for the pain warns of the danger and the man puts on his mask. We may safely say therefore that if a man is killed outright by any gas that this causes less pain and suffering than bullet wounds, while if he becomes a casualty, his sufferings are still less than those of the wounded, and are of shorter duration. A man is out of danger and fairly comfortable forty-eight hours after gassing with phosgene. Even mustard casualties do not require as long hospitalization as most wounds. Do you think the bayonet is more humane than gas? Men are trained to stick it in the belly and twist it around, thus causing painful injury and almost certain death after prolonged agony. Are high explosives and shrapnel humane? Think of the torn and mangled bodies. Remember the cases or pictures you have seen of men with jaws or face shot away. (See plate 1.) In the United States Army, 66 soldiers were totally blinded, and 644 were blinded in one eye by wounds. Gas totally blinded only 4 and partially

BULLET WOUND OF FACE

Showing how extreme may be the deformity produced without being fatal. Compare this result with the use of gas.

blinded 25; 4403 soldiers lost arms or legs; 4790 had the flexibility of one or more joints destroyed. Is not the gas *that does not mutilate*, more humane?

Much disability has been ascribed to gas but the records of the Surgeon General's office and all reports from British, French and German sources indicate that gassing does not increase the liability to tuberculosis, and that there is seldom any permanent serious injury as the result of gassing. The United States hospital records showed that 70,552 gassed cases were treated in hospitals in France. The applicants for war risk benefits claiming that their disability was due to gas have been greatly in excess of that number. Such a serious discrepancy indicates that no faith can be placed in the stories of people who claim to have been disabled as the result of gassing. That many of these men are honest in their belief does not affect the fact.

Still further, gas warfare may be made as humane as desired. Other weapons cannot be so used. Once the bullet or shell has started, all control over it is gone, and the degree of injury produced is a matter of chance. But gas may be varied to suit conditions, and need not necessarily be lethal or produce serious suffering in order to obtain results. Tear gas may be used to disperse a mob with the certainty that the gas will injure no one. The time will come, if it is not already here, when troops or police will be severely criticised for firing on a mob. Tear gas or toxic smokes will disperse any men not provided with the best of masks, yet will cause no casualties. The time may come when rifles, machine guns and explosives will be considered barbarous and inhumane as compared with gas, not only for the control of mobs, but even in warfare.

If all this is true, it disposes of the only logical objection to the use of gas. If at the same time it can be shown that gas is more efficient than other weapons, it will remain in spite of our prejudices.

In discussing the efficiency of gas as compared with other weapons, let us first consider the use of gas against unprotected troops. In the first German attack on April 22, 1915, the total dead were estimated at 5000 and many times that number of casualties. Had the Germans known the effect of the gas they could have walked through to the Channel ports. Similar results were obtained on the Russian front. The total in dead and casualties of three attacks on this front were 140 officers and 21,000 men. Of the total number of casualties reported by the Surgeon General of the United States Army in 1920, 70,552 or 31.49 per cent were gas casualties.

Gas casualties in France—hospital admissions

United States Army

	OFFICERS	ENLISTED MEN			TOTAL	GRAND TOTAL
		White	Colored	Unknown		
Gas, asphyxiating and poisonous, kind not stated	1,249	24,290	526	7,522	32,338	33,587
Chlorine	31	1,607	36	169	1,812	1,843
Mustard gas	883	22,988	515	3,325	26,828	27,711
Phosgene	409	5,540	65	820	6,425	6,834
Arsine	31	345	117	84	546	577
Total	2,603	54,770	1,259	11,920	67,949	70,552

Above does not include deaths on field.

Total of all admissions.....224,089
Admissions due to gunshot.....147,651 or 65.90 per cent
Admissions due to gas..... 70,552 or 31.49 per cent
Deaths in hospital from gunshot wounds... 12,192 or 89.05 per cent
Deaths in hospital from gas..... 1,221 or 8.92 per cent

The number of men dying of each 100 wounded by gunshot missiles was 8.26, by gas 1.73 and by all other military agents 4.72 (Report of the Surgeon General United States Army for 1920, pages 57 and 58).

There is no other known weapon that can produce such effects. In future a nation that should possess the advantage of a gas which penetrates the mask of the enemy, would win the war. The effect of a gas that penetrates the mask, is the same as though the troops were wholly unmasked so far as producing casualties is concerned, with the further result that the morale of the troops may be shattered when they discover that the mask upon which they have been taught to rely, affords no protection. And it must be emphasized that a gas mask quite satisfactory against certain gases may be penetrated readily by gases of a new type.

Moreover these tactical effects can be produced with comparative ease. When using gas it is not necessary to hit your man. Hills, rocks, trees, and even fortifications fail to give adequate protection; trenches and dugouts are actual gas traps. It is no longer necessary to carry strategic positions by assault since the ground may be rendered untenable by mustard or other gases. Relatively few men are required at the front in order to launch a powerful gas attack. Germany, France, Great Britain and the United States had roughly a

total of 507,000 gas casualties. The total number of gas troops employed was about 15,000. These and other facts indicate that gas was probably the most efficient weapon used during the world war. As we have already seen, the most efficient weapons will probably survive.

What may be expected from treaties and agreements not to use gas? While nations may sign such agreements, they cannot rely on them. It cannot be forgotten that Germany broke all treaties and agreements, including her solemn agreement not to use gas.

Germany has not been disarmed in the least so far as chemical warfare is concerned. When the inter-allied commission of chemists visited Germany in order to learn the secrets of her poison gas factories, they were shown the factories that had been manufacturing dyes, perfumes, and medicines for years. It required only a few days or weeks to adapt these plants to the production of poison gases. Generally speaking the same apparatus, retorts, filters, stills, etc., are used in both processes and almost the same raw materials. Germany in 1918 produced 135,000 tons of dye stuffs annually as compared with a total production of 86,000 tons by all the rest of the non teutonic world; and Lefebure[1] in 1920, estimated that Germany was ready at short notice to produce 3000 tons of toxic gas per month. Germany, Russia and other nations are not included in the agreements not to use gas. The chemical industry of Germany was not injured by the war, and the chemical dominance of Germany is a perpetual threat. The Conference of Limitation of Armaments went on record as opposed to the use of gases, but no one of these signatory nations is scrapping its gas program. How can they when gas may be used against them by some power not in the Conference. This Conference did succeed in limiting armaments to a certain extent, and to just this extent it nullified the opposition to the use of gas warfare. For the more armaments are limited, the more powerful in proportion are commercial ships, aeroplanes, and chemical industries, things that cannot be limited or controlled. In this connection Lefebure says,

> A curiously illogical attitude of mind has arisen in certain quarters. There is a tendency among strong adherents to the ideal of world peace to regard themselves as its sole possessors. Every thinking civilian and soldier must adhere to such an ideal; the only point at issue is the method of approaching it.

---

[1] Lefebure. The Riddle of the Rhine, W. Collins Sons & Co., 1921.

The mere fact that a League of Nations is called into being to attain world peace implies recognition of the fact that a definite mechanism and definite measures are required for the purpose; this is self-evident. There are those who, having established their League of Nations, feel that they can attain chemical peace by merely prohibiting chemical war, in other words, they expect their mechanism to achieve its object without functioning, to attain peace by its mere existence. Just as special measures are required to control disarmament in the older branches of warfare, in the same way special measures, but not the same measures, are required to control the chemical peace. Chemical peace guaranteed by a mere signature is no peace at all.

We may conclude therefore that gas warfare is not more brutal or inhumane than other forms of warfare, but it is more efficient than other forms of warfare and will therefore continue to be used; and that the very limitation placed by agreement on armament has increased the importance of chemical warfare relatively because the weapon itself, the ability to make chemicals, cannot be abolished.

If it is even possible that gas may be used against us at some future time, the Medical Department must be prepared to treat gassed cases, with all that this implies with regard to organization, transportation, personnel, supplies, and training of its personnel. The following is written in order to provide officers of the Medical Corps with a brief but comprehensive outline of what they should know concerning chemical warfare and the treatment of gassed cases.

CHAPTER I

Outline History of Chemical Warfare

While certain chemical warfare agents such as Chinese stink-pots and Greek fire are very ancient, our real interest in the subject dates from the introduction of chemical warfare by Germany in the World War. The use of gas was a direct violation of the agreement signed at the Hague Congress in 1899, when all powers represented, except the United States, pledged themselves not to use suffocating or poisonous gases. Admiral Mahan, the American delegate to that Congress refused to commit the United States to the agreement against the use of asphyxiating gas in projectiles for the following reasons:

1. That no shell emitting such gases is, as yet, in practical use or has undergone adequate experiment, consequently a vote taken now would be taken in ignorance of the facts as to whether the results would be of a decisive character or whether injury in excess of that necessary to attain the end of warfare, or immediately disabling the enemy, would be inflicted.
2. The reproach of cruelty and perfidy addressed against the use of these supposed shells was also equally uttered previously against firearms and torpedoes, although both are employed now without scruple.
3. It is illogical and not demonstrably humane to be tender about asphyxiating men with gas, when all are prepared to admit that it is allowable to blow the bottom out of an ironclad at midnight, throwing four or five hundred men into the sea to be choked by water, with scarcely the remotest chance to escape.

The Germans not only violated their agreement when they commenced the use of gas, but they used it foolishly. They used it first on an experimental scale, and not realizing its effectiveness made no effort to follow up its success. Had gas been first used on a large scale, and followed by a general offensive, there is little doubt that the war would have been concluded. The actual result was that the Allies promptly provided protective devices, and began preparations to use gas themselves. Eventually, the Allies sent more gas to the Germans than they received, and at the signing of the Armistice the daily production of toxic gases in America exceeded the total production of toxic gases of all other countries put together. The knowledge of this fact may have influenced the

Germans in signing the Armistice and accepting defeat. It is however an illustration of the futility of depending upon hectic effort in time of war to compensate for the lack of intelligent preparation in time of peace. The accomplishments of the United States in chemical warfare after entering the war were marvelous. For example, the enormous chlorine plant at Edgewood Arsenal capable of a daily production of 80 tons of chlorine was constructed in ninety days. But only a small proportion of American gas ever reached the front. Owing to their previous studies and preparations the Germans maintained the initiative in chemical warfare to the end, and it was only owing to a fortunate series of events that they were not completely successful.

The first gas attack was delivered by the Germans north of Ypres on April 22, 1915 at 4:30 p.m. Chlorine was used from cylinders, one cylinder per meter being installed over a 6 kilometer front. Gas pioneer troops, chemists and meterologists were present. The discharge was five minutes, the wind velocity 2 to 3 miles per hour. The attack, when launched was entirely successful; but the German General Staff knew so little of the weapon that no reserves were at hand to take the ground laid open to them. Professor Haber realized that a tremendous chance for winning the war was lost by the failure to grasp this opportunity, and said that no great weapon could ever have its full strength unless handled by troops and officers who fully understood its possibilities.

Professor Haber, who had conceived this method of attack, was at the front from February until April waiting for favorable weather conditions to launch it. The Allies were unprepared for this attack. It is recorded that they had had information through their military intelligence service that such an attack would be made, but no credence was given to the report. The Allied casualties were about 5000. The second gas attack against the British occurred on April 24 in the Langemarck sector, where there were 1200 casualities in spite of the fact that the British were partially protected by a hastily improvised mask. This mask consisted of a pad saturated with a solution of sodium carbonate and sodium hyposulphite, and 100,000 of these are said to have been produced by the women in England in twenty-four hours after the first attack. Several more attacks against the English and the French were made by the Germans during April and May with partial success, and were continued at intervals during 1915, but by May 24, the British troops

were supplied with the official veil respirator. The greatest cloud attack was in the Champagne in the Fall of 1915, when 24,000 cylinders were released over a 20-kilometer front. The first gas attack by the Allies consisted of a chlorine cloud released from cylinders in the early part of September 1915. It will be seen that five months elapsed before the first use of chlorine by the Germans until the Allies were able to retaliate.

Since protection against chlorine was relatively simple, the Germans introduced phosgene, December 19, 1915, which is more toxic than chlorine, supposing that the Allies could have no protection against it. However, the British Intelligence Service had discovered in advance that the enemy intended to use phosgene and the British Army was protected against it before it was first used by the Germans. But for this circumstance the Germans might have won the war at once. It is easy to understand that the introduction of any toxic gas for which the enemy has no protection would probably have proved decisive then, and with the increased knowledge of the tactics of chemical warfare, it is almost certain that such a gas would decide a war today.

Although clouds of gas released from cylinders had proved very effective, this method was gradually discontinued, because of the great difficulty in carrying out such attacks. The heavy cylinders had to be carried to the front line trench and installed. This means that each cylinder had to be carried by hand, at night, through a difficult system of trenches and traverses, at the expenditure of an enormous amount of labor and effort. As each cylinder would require four men to handle it, the installation of 2000 cylinders on a two mile front, would require more than 8000 men simply as carriers for several days, not to mention the transportation in the rear and the difficult matter of supply. All of this work had to be carried out in such a way as not to interfere with the normal activities of that particular sector, and without making any sound that would advertise this operation to the enemy. The last cloud attack on the British front occurred on August 8, 1916, although there were several such attacks against the French at a somewhat later date.

Both the Germans and the Allies naturally endeavored to develop better methods of delivering gas. The Germans and the French both appreciated the possibilities of the gas shell and developed them. The first German shell containing irritating compounds were used in October and November of 1914. The French retorted promptly

with a lachrymator, and from April, 1915 to July, 1916, lachrymatory shell were used by all parties. The French placed their results at the disposal of the British who rapidly developed a gas shell of their own. The first gas shells contained lachrymators only, but each nationality engaged was constantly developing new types of shells, new fillings, and new tactical uses. Thus in 1916 artillery shells containing toxic gases and various mixtures of toxic and lachrymatory gases were used in increasing numbers by both the Germans and the Allies.

The Livens projector was invented by a British engineer, Lieutenant Livens (afterwards Major). Realizing that clouds of gas were more efficient than shell, he devised a method for throwing gas cylinders into the ranks of the enemy where they would explode, thus releasing a heavy cloud of gas at a time and point where it was totally unexpected. (See plates 2–3.) The projector was simply a tube, or mortar, which could be buried in the ground, filled with a charge that would throw the gas cylinder the required distance. Large numbers of these tubes could be installed at the same time and fired simultaneously by electrical control. In this manner quantities of gas, comparable with those used in cylinders, could be used to produce a cloud which would originate as far as a mile from the point of discharge. The advantages of a cloud attack were secured without dependence on the direction of the wind and with a much greater factor of local surprise. The partially perfected weapon was used by the British in the Arras offensive in April, 1917, and caused many casualties and considerable consternation in the German army. The Germans were not able to retaliate with projectors of their own manufacture until December, 10, 1917.

The Stokes mortar (see plate 4) was also developed by the British for the same purpose. The Stokes mortar fires ordinary artillery shell with great rapidity. Each shell is fitted with an ordinary percussion cartridge and one or more rings of cordite depending upon the range. The soldier simply drops the shell into the muzzle of the mortar. When the shell strikes the firing pin at the bottom of the mortar, the cartridge is exploded, this explodes the cordite and the shell is expelled. It has a range of more than a mile and can be operated as rapidly as shells can be dropped into the mortar.

The Germans reaped the advantage of the next advance in chemical warfare. On the nights of July 12 and 13, 1917, mustard gas

PREPARING BATTERY OF LIVENS PROJECTORS

LIVENS PROJECTORS READY TO FIRE

FIRING BATTERY OF LIVENS PROJECTORS

EXPLOSION OF 14 LIVENS PROJECTORS FIRED AT NIGHT

THREE-INCH STOKES MORTAR

was used against the British at Cambrai. Here again the Germans lost an opportunity to decide the war in their favor, by using the new gas in a small front instead of waiting until they had an ample supply to use on a large scale. Although the masks in use by the Allies afforded good protection against mustard, over 6000 casualties occurred because the British were not familiar with the properties of the gas and failed to use their masks, and because of the vesicant action of the gas for which they had no protection whatever. Mustard was much more toxic even than phosgene, was a persistent gas remaining on the ground for days or even weeks under favorable conditions, and acted as a vesicant on the entire skin area, as well as on the eyes and respiratory tract. From this time on mustard gas was one of the most serious problems of the Allies, and produced nearly eight times more Allied casualties than the various other kinds of German gas. The Allies were obliged to educate all their troops concerning the properties of this new gas, but in spite of all their efforts they suffered a continuous stream of casualties. The chemical composition of this gas was relatively simple and was learned by the Allies almost immediately. Yet owing to the difficulties encountered in making mustard gas in large quantities, it was not until June, 1918, eleven months after the Germans first used mustard, that the French were able to return their first mustard shells. British mustard gas was not in use until September, 1918.

At about the same time that mustard gas was introduced, namely July, 1917, the Germans produced another type of toxic compound in shells, namely the arsenicals. Diphenylchlorarsine, diphenylcyanarsine and other arsenic compounds were used. These compounds caused a violent irritation of the nose and throat, nausea and vomiting. The Germans had expected that the shell burst would create such a fine diffusion of these compounds that the particles would penetrate the respirators mechanically, and that the following irritation and nausea would compel the removal of the masks. If this had succeeded the Allied soldiers would have been helpless against other more lethal gases. Fortunately, this penetration was not secured, since the shell burst did not produce small enough particles. Here again therefore the Germans had the advantage of the initiative and it was the merest good fortune that the Allies were not overwhelmed by any one of these new toxic agents. Thus in following the history of chemical warfare, we

find again and again how great an advantage accrues to the side which first introduces a new chemical agent. It seems almost certain that no nation can neglect chemical warfare to such an extent as to leave it as helpless before a foe as were the British when chlorine was first used. We must at least possess an unbreakable defense against such agents, even though it is eventually decided that we are not to use them ourselves.

DEVELOPMENT OF THE CHEMICAL WARFARE SERVICE

It is easy to understand now that chemical warfare requires a separate department in the army, as it is a distinct and highly technical branch of the service. But on the entry of the United States into the World War there was no chemical warfare service, and several different governmental organizations took up the work. On February 7, 1917, Mr. V. H. Manning, director of the Bureau of Mines, suggested that as that bureau had had considerable experience in connection with mine gases and rescue work, and had a technical staff to work on these problems, the Bureau of Mines would be of use in connection with gas warfare. This suggestion was accepted, and a subcommittee of the National Research Council was formed known as the Committee on Noxious Gases, with Mr. Manning as Chairman. At the first meeting of the committee, April 7, 1917, Mr. Manning was requested to proceed with the organization of a special technical force for the study of chemical warfare problems. The funds for this work were provided by the Bureau of Mines until July 1, 1917, after which funds were provided by the various departments of the Army and Navy. The end result was that the study of chemical warfare in the United States was commenced by the Bureau of Mines.

In May, 1917, the Surgeon General had confidential information that 25,000 troops were to sail for France in June, that they had no gas masks, and this was followed May 16, 1917, by a memorandum from the Adjutant General calling on the Surgeon General to furnish before June, 1918, 1,100,000 respirators, 80,000 trench sprayers, and 1000 oxygen inhalators. As the medical department had no organization for this work, it was turned over to the Bureau of Mines which had already been organized and had commenced operations. Effort was centered upon manufacturing the first 25,000 masks. By enormous effort involving the coöperation of a considerable number of manufacturing firms in the United States,

20,000 gas masks were actually shipped to France before the end of June, 1917. However before they even reached France, it was found that the mask fabric was penetrated by lachrymators, that the nose clips and eye pieces were defective and later it was found that the canister did not afford protection against chlorpicrin. None of these masks could be used.

In addition to this work on the production of gas masks, the Bureau of Mines initiated research work on toxic gases and related subjects. On July 1, 1917, the Bureau had a staff of fifty investigators employed on such problems. Later a number of officers from the Sanitary Corps detailed with the Gas Defense Service, were assigned to this experimental work. Still later as experiments were carried out with offensive material, the Ordnance Department became interested and a number of ordnance officers and enlisted men were assigned. On September 15, 1917, the Bureau of Mines work on gas warfare was moved to the American University, in the outskirts of Washington, and this eventually developed into a large plant equipped for all forms of research on chemical warfare problems. The organization, however, was very defective. Civilians employed by the Bureau of Mines, officers of the Sanitary Corps and from the Ordance Department were all assembled together, with authority and responsibility for the work centering in the Bureau of Mines, a civil organization, instead of in the War Department where such authority naturally belonged. Probably only the intense desire on the part of all concerned to contribute something of value to the American cause enabled this organization to function.

Inasmuch as the Medical Department was directly charged with supplying the A. E. F. with gas defense apparatus it was important to have the manufacture of such equipment directly under its control. Accordingly a certain proportion of the personnel of the newly organized Sanitary Corps was allotted to carry on this work, and on August 31, 1917, all projects of the Medical Department relating to defense against gas were consolidated into the Gas Defense Service. The following quotations from the order effecting this consolidation describe the new organization:

> The duty of providing for the supply of these appliances, of repairing them, and of giving instructions in their use, is performed by a special field service of the Medical Department, known as the Gas Defense Service, the principal

office of which is located in this city (Washington). It comprises three branches: (1) Field Supply Section, (2) Overseas Repair Section, (3) Training Section.

The Field Supply section will purchase or manufacture the appliances named, inspect them, store them, and issue them as needed.

The Overseas Repair section will receive issues made in bulk from the home country, test them, store them, and issue them to troops as required; they will also be charged with the disinfecting and repair of used or injured masks abroad, including all necessary inspections and tests incident thereto.

The Training Section will provide instructions regarding the use of these appliances, the handling of gases used for training purposes, the training of officers and men in the use of gas sampling apparatus, gas detectors, and other means of defense against gases, and will communicate the same to all concerned.

The Overseas Repair section described in this order was later organized and sent to France. The Field Supply Section later became the Gas Defense Division of the Chemical Warfare Service, and the Training Section became the Training Division.

By October, 1917, the problem of the manufacture of gas masks was largely solved by this newly organized Gas Defense Service, and production of American gas masks for issue to troops in France was under way. It is apparent from this brief outline that a large portion of the present Chemical Warfare Service had its inception in the Medical Department. And although it is certain that the duties of gas defense and training, as well as chemical research can be more appropriately carried out in the Chemical Warfare Service than in the Medical Department, it is equally certain that there should be a close liason between the two services, in order to obtain the best results.

THE ORIGIN OF EDGEWOOD ARSENAL

Until October, 1917, it was the duty of the Ordnance Department to provide a supply of toxic gases. It had been intended that the manufacture of such chemical warfare agents should be entrusted to civilian chemical firms, but it was soon found that very few such firms were either able or willing to undertake large scale production of these highly toxic compounds.

It was therefore determined that the government itself should erect and operate such plants. Accordingly the land was purchased near Edgewood, Maryland, October 16, 1917, and actual construction was commenced on November 15, 1917. In spite of many difficulties, construction was so rapid that shells were filled for delivery as early as March 11, 1918, and at the signing of the Armis-

tice, 90 per cent of all the Plants at Edgewood Arsenal had been completed and were in operation.

The extent of this accomplishment may be realized when it is stated that the monthly capacity of these plants was as follows:[1]

Monthly Capacity at Armistice

	tons
Titanium tetrachloride	30
Tin tetrachloride	90
White phosphorus	100
Mustard gas	900
Brombenzylcyanide	90
Phosgene	1050
Chlorpicrin	1500
Gaseous chlorine	1500
Liquid chlorine	895

The gas mask factory and all experimental work on defense problems are also located at Edgewood Arsenal. The future maintainance of Edgewood Arsenal is, therefore, an essential part of preparedness for future conflct, even though this country has no intention of being the first to use gas again.

ORGANIZATION OF THE GAS SERVICE. A. E. F.

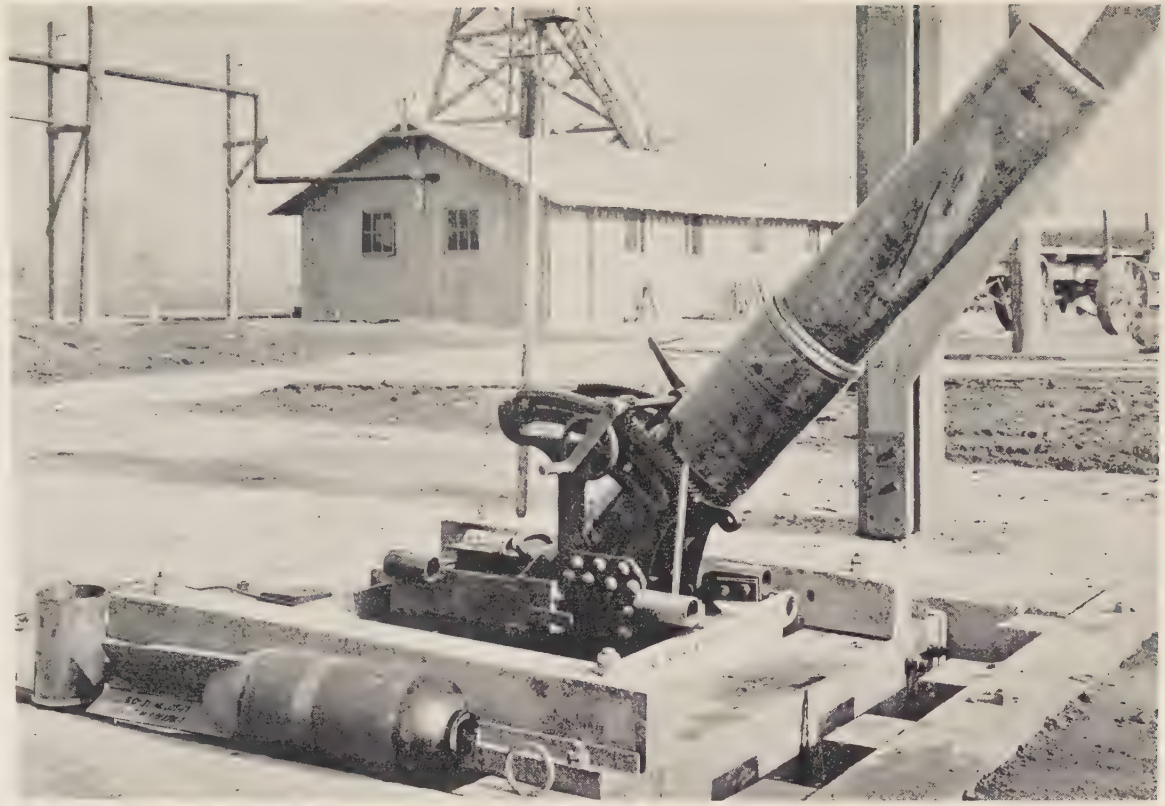
Shortly after the A. E. F. arrived in France, the Gas Service A. E. F. was created by order of the Commander in Chief. Lieut. Col. (now Brig. Gen.) Amos A. Fries was appointed Chief of the Gas Service. The service was organized under the following Divisions: Offensive, Defensive, Technical, Supply and Intelligence. Thus all of these functions were unified and coördinated instead of functioning separately and more or less independently as had been the case with the organization originating in this country. Commencing August 22, 1917, General Fries built up the Chemical Warfare Service A. E. F. until at the time of the Armistice this service consisted of over 600 officers and 3000 men, with a further great expansion planned to fifteen hundred officers and 18,000 men

[1] In 1916 the British output of warfare gas was 5150 tons, in 1917, 18,500 tons, and for ten months of 1918, 15,500 tons. About half of these quantities were liquid chlorine, the remainder being phosgene, chlorpicrin and lachrymators. In 1917 Germany made 2000 tons of gas for shell filling and 1000 tons of chlorine per month. (Official History of the War, Vol. 2, London, 1923.) From this it will be seen that the Edgewood plant alone was capable of producing more gas than Germany made in 1917.

by the summer of 1919. The greater number of enlisted men were on duty with the First Gas Regiment which was organized August 15, 1917, by War Department authority in the United States, but was sent to France early in 1918.

While the foregoing description outlines the genesis of the more important organizations that developed during the war, it by no means describes all of the ramifications. As was natural with a series of organizations that had grown like Topsy, there was a very general lack of coördination in chemical warfare activities in the Spring of 1918. As a result, G. O. 62, War Department, June 28, 1918, created the Chemical Warfare Service of the National Army, which was to include the American University Experiment Station and all organizations of the Army engaged in the problems of Chemical Warfare. In the summer of 1919, Congress passed a bill providing that the Chemical Warfare Service remain in effect until June 30, 1920, and under the Army Reorganization Act of June 4, 1920, the Chemical Warfare Service was created as a separate branch of the Regular Army.

With the creation of the Chemical Warfare Service in 1918, the Medical Department ceased to have any responsibility for gas defense or any other functions of chemical warfare. While the Medical Department has always maintained a close liason with the Chemical Warfare Service through an officer of the Medical Corps detailed for duty on the Staff of the Chief of the Chemical Warfare Service, it soon ceased to take any active part in experimental work. But it has always been recognized that the Medical Department must be responsible for the treatment of men who may be gassed, as well as for many medical problems connected with chemical warfare. Accordingly in October, 1922, the Medical Research Division was organized at Edgewood Arsenal, with the approval of the Chief, Chemical Warfare Service, and the Surgeon General. It was planned that this Medical Research Division should be charged with the duty of studying the pharmacological action of Chemical Warfare agents, of developing the best possible methods for treating gassed cases, and for disseminating this information among the officers of the Medical Corps.



TRENCH MORTAR

CHAPTER II

Physics as Applied to Chemical Warfare

STRUCTURE AND PROPERTIES OF MATTER

Matter consists of an aggregation of small particles called molecules, held together more or less firmly by some attractive force, but the distances between them are very large as compared with their size which is ultra microscopic. Molecules are believed to be constantly moving at very high velocity, and the rate of this motion increases with a rise in temperature. Molecules are not further divisible by mechanical means. A division of the molecule into its component atoms involves a change in the nature of the compound. Roughly speaking the study of the behavior of molecules is physics and the study of the atoms composing molecules is chemistry. While the above statements are theoretical they must approximate the truth very closely because they explain all physical phenomena.

In solids, each molecule appears to be free to move only within a limited space around a central point. The molecules cannot slip by each other. Hence a solid has a constant volume at a constant temperature, and retains its shape irrespective of the position in which it may be placed. In liquids the molecules have greater freedom of motion, and are not fixed in their position in the mass, but may move quite freely past each other, and are constantly changing their relative position. This causes the phenomenon known as *diffusion*. A liquid has a constant volume at a constant temperature, but readily conforms in shape to that of the containing vessel. In the case of a gas, the molecules are still further apart, and have a still greater freedom of movement. The individual molecules are not confined or restricted to any particular space or location, and glide past each other with the utmost freedom. Consequently a given amount of gas has neither a definite volume or a definite shape, but conforms to the size and shape of the confining vessel. The constant and rapid bombardment of the walls of the vessel by the molecules of the confined gas produces the phenomenon of *gas pressure*. A rise in temperature increases the velocity of molecular movement and hence produces an increase in pressure.

Although the molecules of all matter are in constant motion, the molecules of any particular body exert an attractive influence on each other, thus limiting their motion. This attractive force may be called *cohesion.* In solids the molecules are attracted to each other or cohere more or less firmly, but variations in the extent of this cohesion account for various properties of solids such as, brittleness, tenacity and elasticity. In liquids, cohesion is much less pronounced but motion of molecules is not unrestricted. The cohesion of molecules in liquids accounts for the viscosity of liquids, and for *surface tension.* The molecules within the body of a liquid are attracted equally in all directions while those on the surface are only attracted by those below and beside them. Consequently a film is formed upon the surface of a liquid which is under constant tension and tends to contract. Whenever the surface of the liquid is curved as in a drop the tension is perpendicular to the surface and in the direction of the centre of curvature. Surface tension varies greatly with composition. Water has a surface tension of 73 dynes, alcohol 22 dynes and ether 16 dynes. The film formed on the surface of water is sufficiently strong to bear up light insects such as mosquitoes or water spiders.

In gases, the cohesion of the molecues has practically disappeared entirely, and each molecule is ready to move as far as possible from every other molecule, when external obstacles as confining vessels are removed. This accounts for the tendency to unlimited dispersion of gases. The various manifestations of viscosity, fluidity and surface tension of liquids are determining factors in the dispersion of these materials by an explosive charge or other mechanical means. Solids are not usually as finely dispersed by an explosion as are liquids, and liquids of high viscosity and surface tension are not as finely dispersed by a given explosive charge as are liquids of high fluidity and low surface tension. Again just as liquids have a tendency to adhere to the surface of solids with which they have come in contact, so most gases have more or less of a tendency to adhere to or be adsorbed by solids. This tendency increases the more finely the particles of the solid are divided. Charcoal has a remarkable capacity for collecting and retaining a great variety of liquids and gases. Charcoal is very finely porous and presents an enormous surface upon which gases may adhere. This process of condensation of gases or liquids on the surface of a solid material is called *adsorption,* and this property of charcoal is utilized in the gas mask canisters to absorb toxic gases

passing through it. Bunsen recommended freshly precipitated ferric hydroxide as an antidote for arsenic poisoning, supposing that basic ferric arsenite was formed. Recent work indicates that ferric hydroxide in the colloidal state acts by adsorption.

The cohesion between molecules varies inversely with the temperature, that is the lower the temperature the greater the cohesion and vice versa. As temperature increases cohesion decreases to such an extent that a solid melts and becomes liquid and the liquid finally boils and passes into a gas. Water is a solid at temperatures below 0°C. It is a liquid between the temperatures of 0° and 100°C., and above 100°C. it is a gas. All known substances, except such as decompose, can in a similar way by application of the proper temperature and pressure, be obtained as either a solid, a liquid or a gas. The temperature at which any chemically pure substance melts or boils is fixed with such uniformity, that the melting or boiling point when altered proves the presence of impurities in the substance. These critical temperatures (melting point and boiling point) are very important data in chemical warfare. For example, the melting point of dichlordiethylsulphide (mustard) is 13.9°C. or 57°F. This means that pure mustard could not be used in cold weather because it would freeze. Even mustard of 83 per cent purity melts at 8°C. Mustard is often therefore mixed with various substances to prevent freezing. Mustard boils at 219°C., which means that at ordinary temperatures it would vaporize very slowly. It is therefore a persistent compound.

The molecules of a substance move at an average velocity which is definite at a definite temperature. But individual molecules may move at a velocity considerably greater or less than the average, depending upon how they collide with other molecules. When a a molecule near the surface of a solid or liquid acquires a velocity greatly above the average, it overcomes the force of cohesion, and shoots out into the air, and thus becomes a free molecule like that of a gas. It is a well established fact that this process is continuous, for many solids and nearly all liquids are constantly given off to the air a certain amount of their material as gas or vapor. This phenomenon is called *vaporization* or *evaporation*. Evaporation is more rapid the higher the temperature because molecular motion increases with a rise in temperature. For the same reason liquids evaporate much more rapidly than solids. Certain solids vaporize completely without melting, and the vapors on cooling, condense into the solid

state. This phenomenon is called *sublimation.* The vapor tension of such substances becomes equal to or greater than the atmospheric pressure before the melting point of these substances is reached.

If the solid or liquid be placed in a closed vessel so that the molecules that enter the vapor phase are confined, the number of these escaping molecules will gradually increase, and as they accumulate in the atmosphere a number will return to the original body. As the vapor continues to accumulate the number of molecules that return increases until finally an equilibrium will be established for that temperature, when there is a balance between the escaping and returning molecules, and the amount of vapor will not change thereafter unless the temperature changes. The atmosphere is now *saturated* with the gas at the existing temperature. The pressure which such a saturated vapor exerts is called the *vapor pressure.* This vapor pressure is an important property of chemical warfare material since upon this property depends the maximum concentration of any agent that is possible to attain in the field. For example, the vapor pressure of mustard is as follows:

TEMPERATURE	PRESSURE OF MERCURY
°C.	mm.
13.9	0.07
20	0.11
40	0.45
60	1.51
80	4.52
100	11.89

From this the maximum concentration attainable at any temperature may be calculated. The gram molecule (molecular weight expressed in grams, which is 159 in the case of mustard) of any gas occupies 22.4 liters at 0° and 760 mm. pressure. At 40°C. (104°F. or hot summer weather) this volume would be $\frac{.313}{273} \times 22.4$ or 25.68 liters. But at 40°C. mustard has a vapor pressure of only 0.45 mm. instead of 760 mm. Therefore at this temperature a gram molecule of the vapor (159 grams) would occupy $\frac{760}{0.45} \times 25.68$ liters or 43,370 liters. One liter of air saturated with mustard vapor at 40°C. would contain $\frac{159}{43,370} = 0.00366$ gram or 3.66 mgm. of mustard. By com-

paring the maximum concentration attainable with the toxicity of the compound it is possible to determine the value of the compound, so far as these two properties are concerned. For a thirty minute exposure the maximum concentration of 3.66 mgm. is approximately sixty times the minimum lethal dose of mustard.

Owing to this molecular structure, all matter is more or less compressible, but the compressibility of solids and liquids is so small that it may be neglected for practical purposes. In the case of gases whose molecules are widely separated, compressibility is relatively very great.

When a material is heated it expands and it contracts on cooling, probably owing to the effect of temperature on the movement of the molecules which move farther apart when heated. The extent to which various substances expand on heating is different for different substances. The ratio of increase in length of a body, for a rise in temperature of one degree, to the original length of that body is called its *coefficient of linear expansion.* The coefficient of cubical expansion is the ratio of increase in volume per degree to the volume at the original temperature. The coefficient of expansion of mustard is 0.00079 and that of steel is 0.000035. If the temperature should rise 35°, the fractional increase in volume of the mustard is 0.02765 while the fractional increase in interval volume of a steel shell for the same rise in temperature would be 0.001225. Thus we see that the volume of the mustard would increase by 2.76 per cent while the capacity of the shell would increase by only 0.12 per cent, a difference of 2.64 per cent. Owing to this difference in expansion and the incompressibility of liquids, it is evident that if a shell were completely filled with mustard in the winter, and stored until summer a tremendous pressure would be produced and the shell might burst. It is evident that in order that a shell may be filled with mustard at 25°C., and safely heated to a temperature of 60°C., a void of at least 2.64 per cent should be left in the shell.

Most chemical warfare agents are liberated in the field in the gaseous state, and gases are observed to act in certain very definite ways which are expressed as laws:

BOYLE'S LAW

The volume of a gas is inversely proportional to the pressure upon it. If we have a given volume of gas under a certain pressure, and

we double the pressure, the resulting volume will be one-half the original volume. This may be expressed by the equation:

$$pv = p_1v_1$$

where p and v represent any pressure and the corresponding volume, while p_1 and v_1 represent any other pressure and its corresponding volume. This law is readily explained by the molecular structure of the gas. The pressure of a gas is caused by the bombardment of the containing surface by the molecules of the gas. If we decrease the volume one-half, the number of molecules in a given space is doubled, and the number of collisions of the molecules with the containing surface is likewise doubled. The temperature of course is constant.

CHARLES LAW

If a given volume of a gas has its temperature increased the increase in volume is proportional to the original volume and to the rise in temperature, or

$$\frac{V}{T} = \frac{V_1}{T_1} \text{ or } \frac{V}{V_1} = \frac{T}{T_1}$$

where V and V_1 are the two different volumes and T and T_1 the corresponding temperatures expressed in the absolute scale. It has been found that if we start with a definite volume of gas at 0°C. and keeping the pressure constant, slowly raise or lower the temperature, there will be a uniform expansion or contraction of $\frac{1}{273}$ or 0.00366 of the original volume for every variation in temperature of 1°C. Theoretically if we should continue this cooling to − 273°C. and if the same contraction occurred all the way the volume of the gas would have diminished to nothing at this point. This is impossible because all known gases have been found to liquefy long before the temperature of − 273°C. and the gas laws do not apply to liquids. But this change of $\frac{1}{273}$ of the volume of a gas with each degree of temperature has been made the basis of a thermometric scale whose degrees are of the same magnitude as the Centigrade degree but whose zero point is 273° lower than the Centigrade scale. This is known as the absolute scale, and temperatures in the formulae used in expressing the gas laws are expressed in degrees of this absolute scale.

Since according to Boyle's Law the volume varies inversely as the pressure, and according to Charles' Law it varies directly as the temperature, one formula may express both laws as follows:

$$V = \frac{RT}{P} \text{ or } PV = RT$$

where V is the volume, T the absolute temperature, P the pressure and R a new constant depending upon the units employed.

AVOGADRO'S PRINCIPLE

Under the same conditions as to pressure and temperature, all gases contain, in equal volumes, the same number of molecules. This means that the molecules of all gases occupy the same space and therefore the volume occupied by a gas under any given conditions, is dependent only on the number of molecules present irrespective of their size or weight. We find therefore that if we take equal volumes of different gases at the same temperature and pressure, the total weights of these equal volumes of different gases will be in the same ratio as the weights of the individual molecules. It is also found that if we take quantities by weight of two or more different gases which are proportional to their molecular weights, the volumes which these gases will occupy under the same conditions of temperature and pressure will be equal. The molecular weight of any substance expressed in grams is called the *gram molecule* or *mole* of that substance.

Thirty-two grams (molecular weight) of oxygen, 18 grams of water and 71 grams of chlorine will occupy equal volumes in the gaseous state under like conditions of temperature and pressure. It has been found by actual experiment that a gram molecule of any gas under the standard conditions of 0°C. and a pressure of 760 mm. mercury will occupy 22.4 liters. By means of these laws it is possible to calculate the volume which any given weight of gas will occupy under standard conditions and by applying the laws of Boyle and Charles, at any other temperature or pressure. For the volume of a gas is proportional to the number of molecules present, and to the absolute temperature and inversely proportional to the pressure.

$$V = \frac{RNT}{P}$$

where V is volume in liters, T the absolute temperature, P the pressure in millimeters of mercury, N the number of gram molecules, and R is the constant 62.36.[1] Any other units can be used in the same formula providing the constant is changed to correspond.

The ordinary portable gas cylinder used in cloud gas attacks contains 30 pounds of liquid chlorine. What volume will this gas occupy at 25°C. and 740 mm. pressure? 30 pounds = 13,605 grams, and the molecular weight of chlorine being 71, this is equivalent to $\frac{13,605}{71}$ or 191.6 gram molecules. 25°C. = 298 C. on absolute scale.

$$V = \frac{62.36 \times 191.6 \times 298}{740} = 4808.1 \text{ liters}$$

DALTON'S LAW OF PARTIAL PRESSURES

When two gases occupy the same space each exerts the same pressure that it would exert if it occupied the same space alone. This is a natural consequence of Avogadro's hypothesis. Equal numbers of molecules of different gases, when placed in equal volumes will produce equal pressures under standard conditions. That is, the pressure of a gas is proportional to the number of molecules present, irrespective of their magnitude. If then we mix several gases that do not react with each other chemically, each individual gas will exert the same pressure that it would have if it alone were present in the same space and temperature. The total pressure of the mixture will be the sum of the partial pressures of the component gases.

GRAHAM'S LAW OF DIFFUSION

Gases when under uniform conditions diffuse at rates inversely proportional to the square roots of their molecular weights. The pressure of a gas is dependent only on the number of molecules in a given space irrespective of their magnitude. The pressure of a gas is due to the kinetic energy of its molecules, and this kinetic energy is the same for all molecules irrespective of their size or weight. But the kinetic energy of any moving body is equal to half the product of

[1] This constant is derived from 22.4 liters, the space occupied by a gram molecule of gas under standard conditions of 760 mm. pressure and 0°C. or 273° absolute scale, as follows:

$$\frac{22.4 \times 760}{273} = 62.36$$

its weight by the square of the velocity ($\frac{1}{2}$ m. V^2). Hence the only way a small molecule can have as much kinetic energy as a large one, is to have a correspondingly greater velocity. It is clear therefore that the velocity of the molecule must vary inversely as the square root of its mass. Gases do diffuse according to this law, and a gas of low molecular weight will diffuse much more rapidly than a gas of high molecular weight. Thus in the case of hydrocyanic acid (HCN molecular weight 27) and phosgene ($COCl_2$ molecular weight 99), the square root of the atomic weight of cyanogen is slightly more than half the square root of the atomic weight of phosgene. Hydrocyanic acid will therefore diffuse almost twice as fast as phosgene. This fact, taken with the fact that hydrocyanic acid is slightly lighter than air (average molecular weight 28.9), indicates why hydrocyanic acid in small shell did not prove very successful as a warfare gas in spite of its greater toxicity.

CALCULATION OF CONCENTRATIONS

Formerly all data on concentrations of toxic materials in air were expressed in parts per million (p.p.m.). However, this method is applicable only to substances which are gases and not to substances which are liquids or solids. Since most of the present Chemical Warfare Agents are liquids or solids, all concentrations are now expressed in milligrams of toxic material per liter of air. This is the same as ounces per 1000 cubic feet.[2]

Parts per million may be converted to milligrams per liter. In the case of air, or any gas having the same density as air, 1 p.p.m. would be 1 cu. mm. per liter. One liter of air at 0° and 760 mm. weighs 1.293 grams, and 1 cu. mm. of it would weigh 0.0001293 mgm. Hence under standard conditions 1 part per million (1 p.p.m.) of any gas of the same density as air would equal 0.001293 mgm. per liter. The average molecular weight of air is 28.9 and in the case of any other gas, the figure 0.001293 would be changed in proportion to the molecular weight of that gas. Therefore:

$$\text{milligrams per liter} = \frac{M}{28.9} \times 0.001293 \times \frac{P}{760} \times \frac{273}{T} \times \text{parts per million}$$

---

[2] $\frac{1 \text{ ounce}}{1 \text{ cubic foot.}} = \frac{28.35 \text{ grams}}{28.32 \text{ liters}} = \frac{1.001 \text{ grams}}{1 \text{ liter}}$. Since 1 ounce per cubic foot of air is equivalent to 1 gram per liter, 1 ounce per 1000 cubic feet is equivalent to 1 mgm. per liter.

where M is the molecular weight, P the barometric pressure, and the absolute temperature. This may be condensed to the following equations:

$$\text{milligrams per liter} = \frac{M \times P \times 0.0000161 \text{ parts per million}}{T}$$

or the reverse.

$$\text{parts per million} = \frac{\text{milligrams per liter} \times T}{M \times P \times 0.0000161}$$

By these equations parts per million may be converted into milligrams per liter, or vice versa under any condition of temperature or pressure.[3]

Handy equivalents in making computations

1 cm. = 0.3937 inch	1 inch = 2.54 cm.
1 meter = 3.28 feet or 1.093 yard	1 foot = 30.49 cm.
1 kilometer = 0.6213 mile	1 yard = 0.9145 meter
	1 mile = 1.61 kilometer
1 liter = 1.057 quart	1 quart = 0.946 liter
	1 gallon = 3.786 liters
1 cu. meter = 37.27 cubic feet or 1.306 cubic yard	1 cubic foot = 28.32 liters
	1 grain = 64.8 mgm.
1 gram = 15.43 grains	1 ounce = 28.35 grams
1 kgm. = 2.2046 pounds	1 pound = 453.6 grams

$$C = \frac{5(F - 32)}{9} \qquad F = \frac{9C}{5} + 32$$

[3]See appendix for other calculations.

CHAPTER III

Elementary Chemistry of Chemical Warfare

All organic compounds contain carbon. They also contain hydrogen, frequently oxygen, nitrogen and the halogens, and often sulphur, phosphorus, arsenic and other elements. Organic compounds are divided into groups or classes as follows:

1. The aliphatic, in which the carbon atoms in each molecule are arranged in straight or branched chains. This group includes most natural fats and oils and their derivatives.

2. The aromatic or cyclic, in which the carbon atoms of each molecule are arranged in a closed ring to which the atoms of other elements are attached.

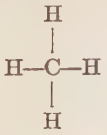
3. The alicyclic, which are cyclic like the aromatic compounds, but they resemble the aliphatic compounds in their properties.

4. The Heterocyclic, in which the molecular structure is cyclic or ring formed, but the ring is not exclusively composed of carbon atoms but may include nitrogen, oxygen, or less frequently other elements.

ALIPHATIC COMPOUNDS

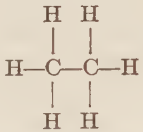
The hydrocarbons. The simplest organic compounds are those composed of carbon and hydrogen, and are called hydrocarbons. From these compounds most of the other compounds of organic chemistry may be derived theoretically and many of them can actually be prepared from the hydrocarbons by various substitutions.

Since carbon has a valence of four, the simplest possible hydrocarbon is CH_4 which exists in nature in coal mines where it is called fire damp, and from decomposition of vegetable matter in marshes, where it is called "marsh gas." Its chemical name is methane. The empirical formula CH_4 corresponds to a structural formula:



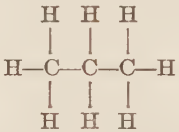
The methane molecule less an H atom is known as the methyl group or radicle (CH_3). The names of the simplest aliphatic hydro-

carbons end in "ane" and the names of the corresponding radicles, the "alkyl" radicles end in "yl." Thus the next higher member of this series is ethane C_2H_6 or



With the loss of one atom of hydrogen, we obtain the ethyl radicle C_2H_5.

Now ethane may be considered as methane CH_4 in which an atom of hydrogen has been replaced by a methyl group. Similarly, if one atom of hydrogen in ethane is replaced by another methyl group, the third member of this series of compounds is obtained. This is known as propane C_3H_8 with the following structural formula:

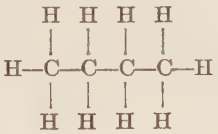


Proceeding in the same way, and substituting methyl groups for hydrogen atoms, we obtain hydrocarbons having four, five or more atoms. The compound containing four carbon atoms is called "butane," and the one containing five carbon atoms, "pentane." The following table gives the names, formulas and some physical

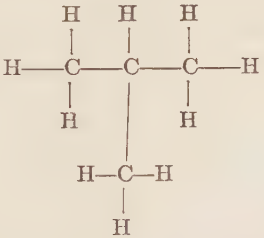
NAME	FORMULA	MELTING POINT	BOILING POINT	SPECIFIC GRAVITY
		°C.	°C.	
Methane	CH_4	−186	−160	0.415 (at −160°)
Ethane	C_2H_6	−172	−93	0.446 (at 0°)
Propane	C_3H_8		−45	0.536 (at 0°)
Butane	C_4H_{10}		1	0.600 (at 0°)
Pentane	C_5H_{12}		36	0.627 (at 20°)
Hexane	C_6H_{14}		69	0.658 (at 20°)
Heptane	C_7H_{16}		98	0.683 (at 20°)
Octane	C_8H_{18}		125	0.702 (at 20°)
Nonane	C_9H_{20}	−51	149	0.718 (at 20°)
Decane	$C_{10}H_{22}$	−31	173	0.730 (at 20°)
Dodecane	$C_{12}H_{26}$	−12	214	0.773 (at m.p.)
Hexadecane	$C_{16}H_{34}$	18	287	0.775 (at m.p.)
Henicosane	$C_{21}H_{44}$	40	215	0.778 (at m.p.)
Hentriacontane	$C_{31}H_{64}$	68	302	0.781 (at m.p.)

properties of this series of compounds. Practically the entire series of compounds from methane up, are contained in petroleum from which they may be obtained by fractional distillation. It will be seen that the molecular constitution of successive members of this series always differs by CH_2. Such a series of compounds, whose members differ by a definite element or group of elements, is called a "homologous series," and the individual members are homologues of each other. It may also be noted that the melting and boiling points and the specific gravity of these compounds increase with the increasing number of carbon atoms in the molecule.

As we have seen, butane is formed from propane by the substitution of a methyl group for one hydrogen atom of propane. But this methyl radicle may be attached to one of the end carbon atoms in the chain in which case we would obtain a straight chain,



whereas, if we substitute the methyl group for one of the hydrogen atoms attached to the middle carbon atoms, we develop a branched chain as follows:

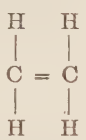


Both of these formulas contain the same number of each kind of elements, and both are expressed by the empirical formula C_4H_{10}. However, they have very different properties and are different compounds. Compounds like these, having the same qualitative and quantitative composition and molecular weight but possessing different properties owing to different arrangements of the atoms in the molecules, are called *isomers*. The isomer containing the straight chain is called normal butane (n-butane) and the isomer with the branched chain is called "iso-butane." This is a general rule for the

nomenclature of straight and branched compounds. It will be readily seen that only two isomers are possible with butane, but as the number of carbon atoms increases in the higher members of the series, a greater number of isomers becomes possible, and many of these isomers are actually known.

From the structural formula of iso-propane, it is apparent that the compound may be regarded as a molecule of ethane in which two hydrogen atoms have been replaced by two methyl groups, and it may be called dimethyl ethane, or it may be regarded as propane in which a methyl group is substituted or methyl propane. These views of chemical constitution of compounds are expressed by constitutional formulas which show what groups of atoms are present in the molecule, though in somewhat less detail than the structural formula. Iso-butane may be written as $(CH_3)_2C_2H_4$ if it is regarded as dimethyl ethane, or by $CH_3-C_3H_7$ if it is regarded as methyl propane.

The unsaturated hydrocarbons. The paraffin series above considered are all saturated, that is, the carbon atoms are united by a single bond. However it is possible for any two adjacent carbon atoms in the molecule to be united by two or even three bonds as in the compounds ethylene C_2H_4 and acetylene C_2H_2.



Ethylene

Acetylene

These compounds are unsaturated, and are much more unstable than the saturated compounds, because they are capable of adding on other atoms or groups of atoms whereas the saturated compounds in which the valence of each carbon atom is satisfied, can only take on other atoms by substitution for atoms already present which must be released.

Unsaturated hydrocarbons containing double bonds are called olefines and those with triple bonds acetylenes. Just as the paraffin series may be regarded as derived from methane by the substitution of hydrogen atoms by methyl groups, so the higher members of the olefines may be regarded as derived from ethylene C_2H_4, and the higher members of the acetylene series as derived from acetylene C_2H_2, by substitution of methyl groups for hydrogen atoms. The

names of the olefines are derived from those of the corresponding saturated hydrocarbons. Thus ethane C_2H_6 yields ethylene C_2H_4, propane C_3H_8 yields propylene C_3H_6 etc. In this series it is also possible to have straight chains or normal compounds and branched chains or iso-compounds. Ethylene, propylene and butylene are gases at ordinary temperatures while the higher members of the series are light oily liquids with progressively higher boiling points. Ethylene is used as one of the materials in the manufacture of mustard. Acetylene is the well known illuminating gas, obtained by the action of calcium carbide on water:

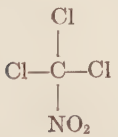
$$CaC_2 + 2HOH = Ca(OH)_2 + C_2H_2$$

When heated to a temperature of about 600°C. the molecules of acetylene combine with each other forming the aromatic cyclic compound benzene C_6H_6. This process by which compounds are formed having the same percentage composition, but whose molecular weights are multiples of the original compound, is called *polymerization*. Benzene is a polymer of acetylene.

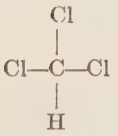
Halogen derivatives of the aliphatic hydrocarbons. The simplest substitution products of the hydrocarbons are their halogen derivatives. Thus we have methyl chloride, CH_3Cl, ethyl bromide, C_2H_5Br, or CH_3-CH_2Br, and propyl iodide C_3H_7I or $CH_3-CH_2-CH_2I$. These compounds are called alkyl halides, and may also be regarded as esters (salts formed with an alcohol as the base), formed by replacing one atom of hydrogen from the corresponding hydrohalogen acid by an alkyl radicle.

When unsaturated hydrocarbons are treated with free halogens, the reaction is at first one of addition, and no by-products are formed until the compound is saturated. Further halogen atoms may then be added by substitution. The alkyl halides present a large number of isomeric compounds.

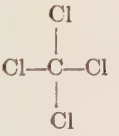
One of the important alkyl halides is trichlor-methane $CHCl_3$ or chloroform.



Chlorpicrin



Chloroform

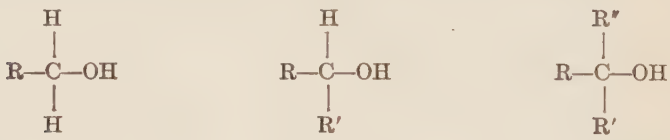


Carbon tetrachloride

When chloroform is treated with more chlorine, tetrachlor methane or carbon tetrachloride results, and chlorpicrin which was largely used as a poison gas during the war is derived from chloroform by replacing the lost hydrogen atom in trichlor methane by a nitro group (NO_2).

Alcohols. All of the alcohols are characterised by the hydroxide groups (−OH) combined with some hydrocarbon radicle. Thus methyl alcohol is CH_3-OH. It may be regarded as methane CH_4 where one atom of hydrogen is replaced by the OH group, or as methyl hydroxide just as NaOH is sodium hydroxide. Ethyl alcohol is C_2H_5-OH. The alcohols may also be regarded as derived from water HOH by the replacement of an atom of hydrogen by an alkyl radicle. However it is only when the carbon atom carrying the OH group is attached either to hydrogen atoms or other open chain carbon atoms (aliphatic hydrocarbons) that the OH group confers the properties of an alcohol. When the OH group is attached to the carbon atoms in the ring of an aromatic compound, the resulting compound is a phenol.

The alcohols are grouped as primary, secondary and tertiary, as the carbon atom carrying the OH group is attached directly to one, two or three other carbon atoms, as:



where R,R′ and R″ represent different alkyl radicles. These different types of alcohols are characterized by the groups

$$-CH_2OH \qquad =CHOH \qquad \equiv COH$$

respectively, and these are called the carbinol groups.

The alcohols react with the alkali metals much as water does with the liberation of hydrogen and the formation of an alcoholate. Thus ethyl alcohol forms sodium ethylate C_2H_5ONa.

$$2C_2H_5OH + 2\ Na = 2C_2H_5ONa + H_2$$

These alcoholates readily react with halogen substituted compounds forming ethers (alkyl oxides). Thus sodium ethylate and ethyl iodide form ordinary ether

$$C_2H_5O\ \boxed{Na + I}\ C_2H_5 = NaI + (C_2H_5)_2O$$

The alcohols readily react with both inorganic and organic acids to form esters or acids in which the replaceable hydrogen is replaced by an alkyl radicle.

When treated with strong dehydrating agents such as sulphuric acid or phosphorus pentoxide, the alcohols may form either ethers or unsaturated hydrocarbons according to conditions. Thus ethyl alcohol may yield ordinary ether $(C_2H_5)_2O$ or at a higher temperature it will yield ethylene C_2H_4. This latter reaction can be carried out by merely heating the alcohol in contact with certain catalytic agents, as aluminium oxide Al_2O_3, Silica SiO_2, china clay or kaolin. This method has been adopted in the large scale production of ethylene in the manufacture of mustard.

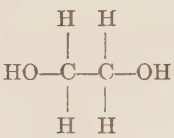
The following table shows some of the characteristics of the lower primary (normal) alcohols.

NAME	FORMULA	MELTING POINT	BOILING POINT	SPECIFIC GRAVITY
		°C.	°C.	
Methyl	CH_3OH		66	0.796
Ethyl	C_2H_5OH	−112	78	0.785
Propyl	C_3H_7OH		97	0.799
Butyl	C_4H_9OH		117	0.805
Amyl	$C_5H_{11}OH$		138	0.826
Hexyl	$C_6H_{13}OH$		157	0.820
Heptyl	$C_7H_{15}OH$		176	0.830
Octyl	$C_8H_{17}OH$		195	0.837
Nonyl	$C_9H_{19}OH$	−5	213	0.834
Decyl	$C_{10}H_{21}OH$	+7	231	0.829

The alcohols can be prepared synthetically by various methods, but in practice, methyl alcohol is obtained from the dry distillation of wood, ethyl alcohol from the fermentation of sugars; and propyl, butyl and amyl alcohols are by-products of this alcoholic fermentation. In distilled liquors they are sometimes called fusel oil. In general, the lower alcohols become more toxic with the increase in carbon atoms. Methyl alcohol is an exception to this rule. The higher alcohols are non toxic because they are comparatively insoluble. These higher alcohols from C_8-C_{18} are found in various plant oils as esters of organic acids. Ceryl alcohol $C_{26}H_{53}OH$ and Myricyl alcohol $C_{30}H_{61}OH$ are constituents of waxes.

It is possible to substitute not only one, but two or more (−OH) groups in the same hydrocarbon molecule, and thus obtain different

alcohols. Those containing but one OH group are called monohydric or monatomic alcohols; those containing two OH groups are called dihydric or diatomic alcohols. Each OH group is attached to a different carbon atom, as molecules containing two OH radicles combined with the same carbon atom are unstable and as soon as formed separate off a molecule of water and become aldehydes. When two OH groups are introduced into the molecule of ethane CH_3-CH_3, the product is glycol, the first member of a series of diatomic alcohols called glycols. Glycol is therefore CH_2OH-CH_2OH, or



Glycerol or glycerine $CH_2OH-CH_2OH-CH_2OH$, is the most familiar member of the triatomic alcohols and is a never failing constituent of all fats where it occurs in combination with the fatty acids. The polyhydric alcohols are not toxic, but are sweet tasting substances whose sweet taste becomes more pronounced as the proportion of OH groups in the molecule increases.

Mercaptans or thio-alcohols. Just as the alcohols may be considered to be derivatives of water by replacing one of the hydrogen atoms by an alkyl group, so the mercaptans are an analogous group derived by replacing one atom of hydrogen sulphide by an alkyl group. They may be prepared by warming alkyl halides with potassium hydrogen sulphide KSH, which is an acid salt of hydrogen sulphide H_2S. All the mercaptans possess obnoxious odors. Butyl mercaptan resembles skunk and was used during the war to produce confusion or mask the use of other chemical warfare agents. It is sometimes mixed with carbon monoxide, illuminating gas, to warn people of its presence when escaping.

Aldehydes and ketones are two classes of compounds which are very closely related. They are both hydrocarbon derivatives containing an oxygen atom united by a double bond to a single carbon atom, and about the only difference between the two compounds is the fact that in the aldehydes the carbon atom carrying the doubly bound oxygen atom is a primary carbon atom (i.e., is united directly to only one other carbon atom) and hence must have one other bond linked to a hydrogen atom; while in the ketones the carbon atom carrying the

doubly bound oxygen is a secondary carbon atom, that is it is in direct union with two other carbon atoms. Therefore in aldehydes the carbon with its doubly bound oxygen atom is at the end of the chain of carbon atoms, while in ketones, it is within the chain between two carbon atoms. Hence the characteristic group of the aldehydes is $-C\overset{O}{\overset{\|}{\underset{H}{\diagdown}}}$ while that of the ketones is $>C=O$.

The names of the aldehydes are derived from those of the acids that are formed from them by oxidation. Formaldehyde on oxidation yields formic acid; acetaldehyde yields acetic acid. The ketones are named from the hydrocarbon radicles to which the keto group $=C=O$ is combined. Thus $CH_3-CO-CH_3$ is di-methyl ketone, and is commonly called acetone. $CH_3-CO-C_2H_5$ is called methyl ethyl ketone, etc. Thus it is seen that aldehydes and ketones possess certain characteristics in common since both contain the carbonyl group CO.

The oxidation of primary alcohols yields aldehydes

$$2CH_3-CH_2OH + O_2 = 2CH_3-CHO + 2H_2O$$

The oxidation of secondary alcohols yields ketones

$$2CH_3-CHOH-CH_3 + O_2 = 2(CH_3)_2\,CO + 2H_2O$$

Both aldehydes and ketones may be reduced by hydrogen with the formation of alcohols, the aldehydes forming primary and the ketones secondary alcohols.

Aldehydes and ketones can both be oxidized with the production of acids; also by virtue of the double bond between the oxygen and carbon atoms of the carbonyl groups, aldehydes and ketones are both capable of adding on certain compounds directly or without substitution. Both add on hydrocyanic acid. Aldehydes add on ammonia. Thus with ammonia, formaldehyde forms a crystalline compound hexamethylene-tetramine, commonly called urotropin $(CH_2)_6,N_4$. This material, which readily gives up ammonia, was used in gas masks early in the war under the name of "hexamine" to protect against phosgene.

Aldehydes are unstable compounds, oxidizing readily, and when treated with alkaline hydroxides they polymerize.

The most important ketone is dimethyl ketone $CH_3-CO-CH_3$ or ordinary acetone. Bromacetone $CH_3-CO-CH_2Br$ has been used as a lachrymator.

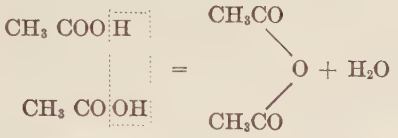
Acids. As has been stated, aldehydes can be further oxidized to acids. These acids are characterized by the group $-C\begin{matrix}O\\ \diagdown\\ OH\end{matrix}$, which is called carboxyl. The acids derived from the aliphatic hydrocarbons are usually called fatty acids because they occur as esters of glycerol in fats. The lower members of the series are liquids, are soluble in water and are fairly strong acids, though weaker than inorganic acids. The higher members are fat like solids. They are named from the hydrocarbons from which they are theoretically derived as follows:

NAME	FORMULA	MELTING POINT	BOILING POINT	SPECIFIC GRAVITY
		°C.	°C.	
Formic	HC OCH	8.3	101	1.2310
Acetic	CH_3COOH	16.6	118	1.0515
Propionic	C_2H_5COOH	−36.0	141	0.9985
Butyric	C_3H_7COOH	−2.0	163	0.9599
Valeric	C_4H_9COOH	−58.5	186	0.9560
Caproic	$C_5H_{11}COOH$	−1.5	205	0.9450
Heptylic	$C_6H_{13}COOH$	−10.5	223	0.9186
Caprylic	$C_7H_{15}COOH$	16.5	237	0.9100
Monylic	$C_8H_{17}COOH$	12.5	254	0.9110
Capric	$C_9H_{19}COOH$	31.4	269	
Palmitic	$C_{15}H_{31}COOH$	62.6	271	0 8527
Margaric	$C_{16}H_{33}COOH$	60.0		
Stearic	$C_{17}H_{35}COOH$	69.3	291	0.8454

The fatty acids may contain one or more carboxyl groups in the same molecule, usually attached to different carbon atoms. Those acids containing only one carboxyl group are called monobasic acids because they are capable of neutralizing only one equivalent of a base. Those acids containing two carboxyl radicles are called dibasic acids. The simplest dibasic acid is oxalic acid $C_2H_2O_4$ or

$$\begin{matrix}COOH\\ |\\ COOH\end{matrix}$$

Anhydrides. When a molecule of water is removed from two carboxyl groups an anhydride is formed, thus



Acetic acid Acetic anhydride

Corresponding anhydrides of the other fatty acids are known. The lower members are liquids, the higher members, solids. The lower members readily react with water regenerating the acids from which they were formed.

When an organic acid is acted upon by a phosphorus halide, a halogen atom replaces a hydroxyl atom in the carboxyl group

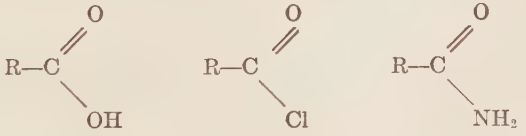
$$3CH_3 - COOH + PCl_3 = 3CH_3COCl + H_3PO_3$$

Acetic acid Acetyl chloride

The names of these compounds are derived from their corresponding acids by changing the termination ic to yl. Thus propionic acid C_2H_5-COOH yields propionyl chloride C_2H_5-COCl, etc. These compounds are commonly called *acyl halides.* The acyl halides react with alcohols to form esters. When an acyl halide is treated with ammonia, the halogen is replaced by the $-NH_2$ or amido group.

$$CH_3COCl + NH_3 = CH_3CO - NH_2 + HCl$$

The product so formed is called an *acid amide,* in this case acetamide. The acid amides may be regarded as organic acids in which the hydroxyl of the carboxyl group is replaced by the amino group $-NH_2$.

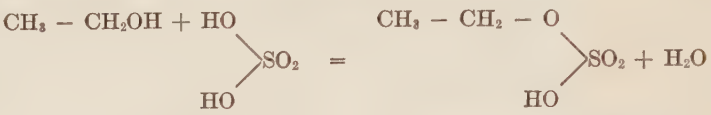


Acid Acyl chloride Acid amide

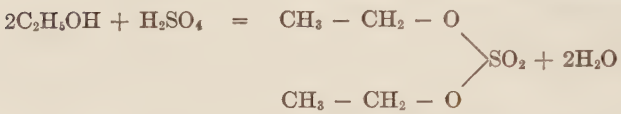
Esters are formed by replacing the hydrogen of an acid by an alcohol

$$C_2H_5OH + CH_3 - COOH = CH_3 - COOC_2H_5 + H_2O$$

In the case of acids having two replaceable hydrogen atoms as sulphuric acid an acid ester or a neutral ester may be formed depending on whether one or both hydrogen atoms are replaced.



Alcohol — Ethyl hydrogen or acid sulphate



Diethyl sulphate (neutral)

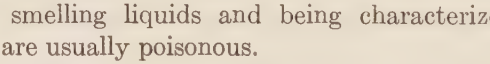
Similarly ethyl nitrate is formed from nitric acid. The ester of the polyhydric alcohol glycerine with nitric acid, is the well known explosive commonly called nitro glycerine. Nitrocellulose is a similar compound, cellulose being related in structure to the alcohols. The esters of the organic acids are found in fruits, and are used in making artificial fruit juices. The glyceryl esters of the higher fatty acids are fats, and the esters of other higher alcohols occur as waxes.

The nitriles are alkyl cyanides as CH_3CN and C_2H_5CN or esters of hydrocyanic acid HCN. They are usually prepared by the action of an alkyl halide on potassium cyanide.

Methyl cyanide

They are called nitriles because they are hydrolyzed by water with the formation of an organic acid and ammonia. Methyl cyanide yields acetic acid and is called aceto-nitrile. The nitriles are usually pleasant smelling liquids and being characterized by the group $-C\equiv N$ are usually poisonous.

If the alkyl halide is treated with silver cyanide AgCN instead of potassium cyanide, the product contains the group $-N\equiv C$ and is called an iso-nitrile. Thus



Methyl chloride — Methyl-isocyanide

Another group of esters are the iso-thiocyanates, the esters of iso-thiocyanic acid $HN=C=S$. These compounds are called

mustard oils. Allyl iso-thiocyanate, $CH_2=CH-CH_2NCS$, is the natural oil of black mustard. It is a heavy oily liquid of a pungent odor, boiling at 151°. It produces blisters when placed on the skin but must not be confused with mustard gas which is $(ClC_2H_4)_2S$ a very different compound. The latter was called mustard because it was somewhat similar in odor and general properties to true mustard, allyl iso-thiocyanate.

Ethers. The alcohols may be regarded as alkyl hydroxides derived from water, and the ethers are the alkyl oxides

HOH	C_2H_5OH	$C_2H_5OC_2H_5O$
Water	Alcohol	Ether

They may also be regarded as two molecules of alcohol with the loss of a molecule of water.

$$CH_3\,OH + H\,OCH_3 = CH_3 - O - CH_3$$

Methyl ether

The two alkyl radicles attached to oxygen may be alike or they may be different. $CH_3OC_2H_5$ is methyl-ethyl-ether. The ethers are named for the hydrocarbon radicles they contain.

The ethers are liquids boiling at lower temperature than the corresponding alcohols. They may be prepared by heating an alcohol with sulphuric acid. The compounds called thio-ethers are alkyl sulphides. They are like the ethers except that they contain a molecule of sulphur instead of oxygen, thus bearing the same relation to the ethers, as the mercaptans to the alcohols. They may be prepared by treating the alkyl halides with sodium or potassium sulphide

$$2C_2H_5Cl + Na_2S = 2NaCl + (C_2H_5)_2S$$

Diethyl sulphide

Mustard gas is diethyl sulphide in which chlorine is substituted in the alkyl groups.

$$\begin{array}{l} ClCH_2 - CH_2 \\ \qquad\qquad\quad \diagdown \\ \qquad\qquad\quad\ \ S \\ \qquad\qquad\quad \diagup \\ ClCH_2 - CH_2 \end{array}$$

The amines. When the hydrogen atoms of ammonia are replaced by alkyl groups amines are formed. The amines are primary,

secondary or tertiary depending upon whether one, two or three hydrogen atoms of ammonia are replaced by alkyl groups,

$CH_3 - NH_2$	$(CH_3)_2 = NH$	$(CH_3)_3 \equiv N$
Methyl amine (primary)	Dimethyl amine (secondary)	Trimethyl amine (tertiary)

The characteristic group of the primary amines is $-NH_2$, of the secondary amines $=NH$ and of the tertiary amines $\equiv N$. The amines are stronger bases than ammonia, and combine with acids to form salts. The basic properties increase with the number of alkyl groups present. They may be prepared from ammonia and an alkyl halide $C_2H_5Cl + NH_3 = C_2H_5NH_2 + HCl$.

With water they react to form hydroxides

$$(CH_3)_2{=}NH + H_2O = (CH_3)_2{=}NH_2OH$$

Dimethyl amine — Dimethyl ammonium hydroxide

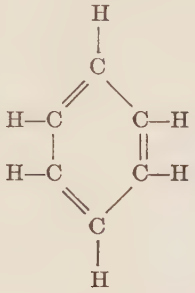
(When instead of ammonia, the amino group NH_2 is substituted for the hydrogen of acids, substances called amino acids are formed. The proteins are composed by the combination of a number of amino acids.)

When the hydrogen atoms of phosphine PH_3 or arsine AsH_3 are replaced by alkyl groups, *phosphines* or *arsines* are formed. Thus they are similar in structure to the amines but phosphorus or arsenic takes the place of nitrogen. Many derivatives of the arsines have been used in Chemical Warfare. Diphenylchlorarsine and diphenylcyanarsine were introduced by the Germans in the so-called blue cross shell.

Carbohydrates contain carbon with hydrogen and oxygen in the proportion to form water $C_n(H_2O)_n$. They are in the nature of aldehyde or ketone alcohols, for they all contain OH groups, and most of them contain the aldehyde group CHO or the ketone group CO. The carbohydrates include the sugars, starches, celluloses, gums, pectins, etc. Most of the higher carbohydrates are hydrolyzed by ferments or acids yielding the lower members.

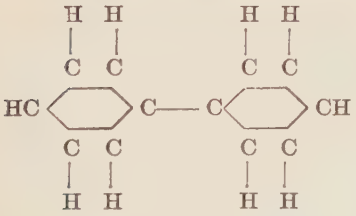
Glucosides are compounds occurring in plants containing a sugar, commonly glucose, united with an aromatic alcohol, aldehyde, acid or other compound. Many of them are toxic. The action of digitalis and many other drugs is due to the glucosides. Amygdalin, the glucoside of bitter almonds, is composed of benzaldehyde, hydrocyanic acid and glucose.

Aromatic hydrocarbons. The number of carbon atoms in a ring is generally six, any other arrangement being much less stable. The simplest possible aromatic hydrocarbon is benzene C_6H_6. Since carbon is tetravalent and hydrogen is monovalent, various diagrammatic schemes have been devised to account for the extra valences of carbon. The following is most popular.

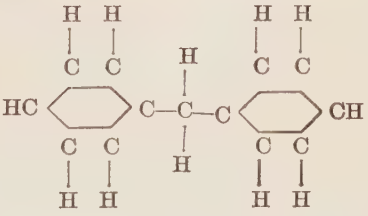


An entire series of hydrocarbons may be derived from benzene by replacing one or more of the hydrogen atoms with alkyl groups, which thus form side chains from the ring or nucleus. Thus the addition of a methyl group yields toluene $C_6H_5 = CH_3$, and two methyl groups yields xylene $C_6H_4 = (CH_3)_2$. The radicle C_6H_5 is called phenyl.

It is possible to have two or more phenyl groups attached to each other either directly or through one or more other carbon atoms.

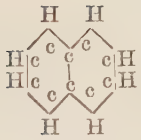


Diphenyl $C_6H_5 - C_6H_5$



Diphenylmethane $C_6H_5 - CH_2 - C_6H_5$

The aromatic hydrocarbons with many of their derivatives and some aliphatic compounds are found in coal tar. The lower members of the benzene series are volatile liquids, while the higher members and all the members of the naphthalene series and other polynuclear hydrocarbons, are solids with high boiling points.



Napthalene $C_{10}H_8$

Anthracene $C_{14}H_{10}$

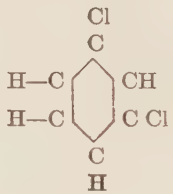
Phenanthracene $C_{14}H_{10}$

They are insoluble in water, soluble in most organic solvents, and are themselves good solvents. Chemically, they are usually more active than the aliphatic hydrocarbons, since the ring is unsaturated.

When there are two or more atoms or groups attached to the benzene ring different products are obtained depending upon the relative positions occupied by the substituting atoms or groups.

To designate these positions the carbon atoms are numbered in a clockwise direction beginning with one of the substituents. The relative position of two substituents is indicated by the term, "ortho" "meta" or "para"; ortho positions being on adjacent carbon atoms, para position on opposite carbon atoms and meta positions intermediate between the other two.

1, 2 ortho position
1, 3 meta position
1, 4 para position



1, 3 dichlor benzene or meta dichlor benzene

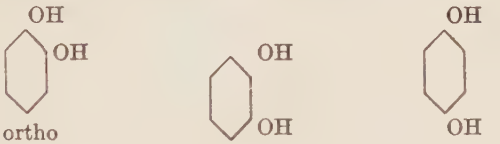
When a group is introduced into an organic compound, the position it will take will depend upon the nature of the substituent, the method and conditions of introducing it, and upon the nature and position of other substituents already within the molecule. If chlorine is introduced into methyl benzene or toluene, it will enter the positions para and ortho to the methyl group, but if the compound contains a carboxyl group as benzoic acid C_6H_5COOH, the chlorine will take the position meta to the carboxyl.

When a compound containing a side chain, like toluene $C_6H_5CH_3$, is treated with a halogen, the substitution may take place either in the ring or in the side chain, and the two compounds will have quite different properties, though having the same empirical formula C_7H_7Cl. Compounds like benzyl chloride $CH_2Cl-C_6H_5$ with the

halogen in the side chain resemble the aliphatic halogen compounds in their properties, and the halogen is readily replaced by other groups. These compounds are powerful lachrymatory agents especially the bromine and iodine derivatives. But when the halogen is substituted in the ring, the compound is much more stable and the halogen is replaced by other groups with greater difficulty.

The phenols are compounds in which one or more $-OH$ groups are attached directly to the nucleus of the aromatic hydrocarbon. When $-OH$ was substituted in the aliphatic compounds it conferred the properties of alcohols. But $-OH$ in the aromatic radicle gives acidic properties. Phenol C_6H_5OH, the simplest compound of this series, turns litmus red and will neutralize bases to form salts. Cresol $CH_3-C_6H_4-OH$ occurs in ortho, meta, and para forms, mixtures of which are called tricresol. Two or three $-OH$ groups may be substituted, when we have dihydroxy benzenes or trihydroxy benzenes.

The three following isomeric dihydroxy benzenes occur:



Pyro chatechin or catechol — Resorcinol — Hydroquinone

Of the trihydroxy benzenes, pyrogallol, 1, 2, 3, trihydroxy benzene and phloroglucin 1, 3, 5, trihydroxy benzene are well known.

Should the OH group be introduced into the side chain instead of the ring we have essentially an aliphatic compound or alcohol. $CH_2OH-C_6H_5$ is derived from toluene $CH_3-C_6H_5$, possesses the properties of an alcohol and is named benzyl alcohol. It is recommended in ointment form to relieve itching caused by various skin irritants. Benzyl alcohol is isomeric with cresol $CH_3-C_6H_4OH$.

The sulphonic acids are compounds containing the group $-SO_3H$ attached to a hydrocarbon radicle. Theoretically it might be combined with either the side chain or the nucleus, but practically it is combined only with the nucleus. They are obtained by the action of sulphuric acid on the hydrocarbon and the process is called sulphonation, and the hydrocarbon is said to be sulphonated. The sulphonic acids are solids which cannot be melted or volatilized without decomposition but most of them are soluble.

Nitro compounds. Nitric acid also acts directly on aromatic hydrocarbons replacing one or more hydrogen atoms with the nitro group $-NO_2$. This process is called nitration. When benzene is acted on by nitric acid, nitro benzene is first produced $C_6H_6 + HONO_2 = C_6H_5 - NO_2 + H_2O$. By the action of more nitric acid, other nitro groups are added on, producing dinitrobenzene $C_6H_4(NO_2)_2$, trinitrobenzene $C_6H_3(NO_2)_3$, etc. Similar compounds may be formed from other aromatic compounds. Thus we have nitrotoluene, $CH_3-C_6H_4NO_2$, dinitro toluene $CH_3-C_6H_3(NO_2)_2$ and trinitro toluene $CH_3-C_6H_2(NO_2)_3$ which is used as the explosive commonly called T N T. The more highly nitrated derivations are often highly explosive. Thus we have picric acid which is trinitro phenol $HOC_6H_2(NO_2)_3$. Most of these compounds are toxic, and munition workers often suffer from this intoxication.

Aromatic amines are characterized by the addition of the amino group $-NH_2$, which is attached to the nucleus or ring. They are commonly made by reducing the corresponding nitro compounds with hydrogen or other reducing agent. Thus nitro-benzene yields aniline $C_6H_5NH_2$, the simplest aromatic amine, and nitronaphthalene $C_{10}H_7NO_2$ yields naphthalamine $C_{10}H_7NH_2$. The aromatic amines are basic and form salts with acids, but they are less basic than the aliphatic amines. They are used especially in the manufacture of dyes.

Diazo or diazonium compounds. These are aromatic hydrocarbons carrying the $-N_2=$ group. It is not known how the two atoms of nitrogen are attached. The following formulas are given

	—N—
—N = N—	N
diazo group	diazonium group

These compounds are usually very unstable. They are much used in the manufacture of dyes.

Other compounds such as the aromatic carboxylic acids, containing the group $-COOH$, the acid amides with the group $-CONH_2$, the esters, nitriles, aldehydes, ketones, etc., may similarly be formed from the aromatic hydrocarbons and are similar in many respects to the corresponding aliphatic compounds. The most important aromatic aldehyde is benzaldehyde C_6H_5CHO, a component of oil of bitter almonds. It is a colorless liquid having a strong odor and

taste of almonds and boiling at 170°C. The aromatic ketones contain the keto group = CO combined with an aromatic hydrocarbon. The aromatic ketones resemble the aliphatic ketones in general characteristics. The simplest aromatic ketone is diphenyl ketone $(C_6H_5)_2CO$ or benzophone. The simplest mixed aromatic-aliphatic ketone is aceto phenone $C_6H_5-CO-CH_3$ or phenyl methyl ketone. When this is chlorinated chloracetophenone $C_6H_5-CO-CH_2Cl$ is formed. This is a very powerful lachrymator which will undoubtedly be used extensively in future because although not the most powerful lachrymator known, it may be manufactured readily and cheaply.

The terpenes. Many of the volatile oils of plants are esters, but the so-called essential oils are terpenes, i.e., oil of turpentine, oil of lemons etc. Turpentine is a mixture of terpenes. When distilled the lighter members pass over leaving resin or colophony. Most of the Terpenes correspond to the formula $C_{10}H_{16}$. Terpenes readily polymerize and oxidize, both processes yielding resins. They combine with the halogens forming addition compounds that are frequently crystalline. The cholesterols are closely related to the terpenes. They contain an OH group and are secondary alcohols. Their structure is not thoroughly known.

Alkaloids are the basic substances occurring in plants which contain in their constitution either a pyridine, quinoline, isoquinoline or pyrol rings. These are all aromatic compounds. Quinoline is C_9H_7N.

Quinoline

Iso-quinoline

The following charts present the fundamental relations of organic compounds. In the aliphatic series chart, it is possible to pass from ethane to every other compound indicated, by following the arrows. The reagent required to effect each change, appears upon the arrow showing that change. The chart includes methods for passing up and down the series.

In the aromatic series chart, it is possible to pass from benzene to every other compound shown. As all derivatives could not be shown, the chart is limited essentially to the monosubstitution products of benzene. A simplified substitution table is included.

CHAPTER IV

METEOROLOGY

Meteorology is the study of atmospheric phenomena. The condition of the atmosphere, its changes in condition, and the causes of these changes have a direct bearing on the dispersion of Chemical Warfare material.

The atmosphere is the envelope of gas surrounding the earth, and has all the physical properties of gases. One cubic centimeter of pure dry air under standard conditions weighs 0.0012927 gram; 13 cubic feet weigh one pound.

The four major constituents of pure dry air are as follows:

	N.	O.	ARGON	CO_2
Volume per cent	78.04	20.99	0.94	0.03
Weight, per cent	75.46	23.19	1.30	0.05
Atomic weight	14.04	16.00	39.9	

Owing to constant diffusion the composition of atmospheric air is remarkably constant. The following table gives the per cent composition of inhaled and exhaled air:

	INHALED	EXHALED
N	78.04	78.04
O	20.99	16.03
Argon	0.94	0.94
CO_2	0.03	4.40

The CO_2 exhaled by animals is largely consumed by vegetation, so that the balance between O and CO_2 is practically stable. If CO_2 increased greatly, vegetation would flourish while animal life would decline.

The amount of water vapor is small and never exceeds 4 per cent, but it is a most important constituent.

Air also contains organic particles, bacteria, and spores of plants. In city streets these may amount to 3000 per cubic meter. The inorganic particles are called dust. In a city, 100,000 dust particles

per cubic centimeter is an ordinary count. There is less dust at higher elevations and over the ocean it may vary from 500 to 2000 particles per cubic centimeter. Dust is the chief cause of haze, serves as centers of condensation for fog and raindrops, and is the cause of sunset and sunrise colors and twilight.

PRESSURE OF THE ATMOSPHERE

This is simply the weight of the column of air above the earth's surface. The average pressure is 14.7 pounds per square inch, but it is usually measured by the barometer in millimeters of mercury that may be balanced by the weight or pressure of the air. The pressure varies with altitude above sea level, moisture in the atmosphere, and other factors. The pressure varies daily, annually and irregularly. The daily variation, never amounting to more than 0.2 inch, is usually small. The chief maximum usually occurs about 10:00 a.m. and the chief minimum at 4:00 p.m. A secondary maximum occurs at 10:00 p.m. and a secondary minimum at 4:00 a.m. The pressure is greater in summer and less in winter; this variation again does not ordinarily amount to more than a few tenths of an inch. It varies also with latitude, being highest at the equator and growing steadily less with higher latitudes.

CONSTRUCTION OF ISOBARIC CHARTS

These are constructed by making use of the normals of pressure which have been determined for various stations in all parts of the world. These normals of pressure are first reduced to sea level and are then charted on a map, and lines are drawn connecting those stations having the same value. These lines are called isobars.

The irregular variations in pressure, especially in temperate zones are far larger than any periodic variations. Variations in pressure of an inch or two follow each other in rapid succession.

Since a gas tends to expand and occupy all space open to it, no theoretical limit can be set to the height of the atmosphere. However the atmosphere cannot extend more than 21,000 miles and turn with the rotation of the earth. At this distance centrifugal force and gravitational attraction balance so that the air would be abandoned. The middle layer of the atmosphere is at a height of 3.6 miles, i.e., there is as much air above as below this level.

HEATING AND COOLING OF THE ATMOSPHERE

Practically all atmospheric heat comes from the sun. The heat from all other sources is not sufficient to change atmospheric temperatures 0.25°F. The sun is constantly emitting 140,000 horse power of energy, of which the earth receives only one two-billionth part, yet the energy received during a year would melt a layer of ice 241 feet thick covering the entire earth. The amount of insolation varies with the nearness to the sun, the directness of the rays and the duration of the rays. These variations all depend upon the revolution of the earth about the sun, and the inclination of the axis of the earth in its daily revolution. The earth is nearly 3,000,000 miles nearer the sun on January 1 than on July 1 and receives 7 per cent more insolation in January. The greater heat of July is due to the fact that in July the north pole is turned toward the sun and the rays are received more directly on the upper hemisphere. On December 21 the north pole is turned away from the sun, and it is winter north of the equator and summer south of the equator. Thus insolation and temperature vary with the latitude and the time of the year.

EFFECT OF INSOLATION ON THE OCEAN

The ocean reflects about 40 per cent of the insolation it receives, and transmits the remainder to considerable depths. However the ocean changes little in temperature between day and night, because (1) 40 per cent of insolation is reflected and lost, (2) a considerable part of heat received is used in evaporation, (3) it requires more heat to raise the temperature of a given quantity of water than any other substance, (4) the heat is transmitted and mixed by convection to vast depths, so that the surface layer practically does not change. Therefore the temperature change of the ocean between day and night is very small, never amounting to more than one or two degrees.

EFFECT OF INSOLATION ON THE LAND

Dry ground reflects but a few per cent of insolation, transmits practically none, and thus absorbs nearly all. The rise in ground temperature under insolation is very great. If ground is wet or covered with vegetation the change in temperature between day and night is much less than for dry ground, but is still considerable and always greater than for the ocean.

EFFECT OF INSOLATION ON THE ATMOSPHERE

Pure air reflects and absorbs but a minute quantity of insolation and transmits practically all. The temperature of the air therefore changes little between day and night.

During the day, both in summer and winter, the temperature of the ground is much higher than the temperature of the air. The summer difference is two or three times the winter difference. During the night the ground is slightly colder than the air in winter, and slightly warmer in summer. The air is slightly warmer than the ocean by day and slightly cooler by night. The ocean can play no part in heating the atmosphere by day or cooling it by night.

The air and the ground are both poor conductors of heat. The ground may become very hot during the day, but being a poor conductor the heat does not penetrate more than 2 or 3 feet. At night the ground cools off rapidly due to radiation. The air can remain in contact with ground many degrees hotter than itself for a whole day, yet by conduction alone, only the lower two or three feet would become perceptibly heated. However, convection or the transference of heat from one point to another by means of the circulation of the liquid or gas itself, takes place in liquids or gases alone. Convection is primarily due to the fact that the liquid or gas, when heated expands, and becomes lighter than the cooler portions, and therefore rises. Since the atmosphere is gaseous, convection will occur if it is heated in one place more than another. The equator is constantly heated more than the polar regions; the convectional currents thereby set up constitute the trade winds. The continents are warmer than the adjacent oceans during the summer and colder in winter. There should then be a circulation between the continents and the ocean; this is the cause of the monsoons. The land is heated more than the adjacent ocean during the day, while at night the land is colder than the adjoining ocean. This is the cause of the land and sea breeze.

If, however, a given quantity of gas (air) becomes heated, it rises, and arrives at regions where the pressure is less. It therefore expands and grows cooler even if conduction and radiation have no effect upon it. These temperature changes are called adiabatic because they take place without the interchange of heat with the surroundings. The amount of such cooling for air is 1.6°F. for a rise of 300 feet, or 1°C. for 100 meters. This assumes that the air remains unsaturated with moisture. Thus if a cubic foot of air rises 3000 feet it cools 16

degrees without gaining or losing heat to its surroundings. Similarly if the same air descends and becomes compressed, a corresponding rise in temperature takes place. If such rising air contains moisture, it may be cooled to the point (dewpoint) when this moisture will be precipitated. The cumulus clouds, which are masses of white cloud looking like exploded cotton bales, usually with rounded dome like summits and flat bases, are simply the heads of rising air columns. A pocket of warm moist air rises, expands and is cooled. If the rise is to a sufficient height, it may become cooled to such a degree that it can no longer hold the moisture which condenses in the form of cloud. These clouds have flat bases because the pockets of rising air have approximately the same temperature and moisture over considerable areas, and reach the stage when they can no longer hold moisture at about the same height.

VERTICAL TEMPERATURE GRADIENT

The temperature is different at different heights above the earth's surface. This change in temperature with elevation is called the vertical temperature gradient. It varies with the time of day, year, and type of weather, but its average value for all times and places is considered to be 1°F. for 300 feet elevation or 0.6°C. for 100 meters. This statement only applies to the atmosphere to a height of two or or three miles. Beyond this point the vertical temperature gradient is more regular, and its value is somewhat greater than near the earth's surface. After a height of six miles or more, the temperature appears to remain constant with elevation. This layer of constant temperature is called the iso-thermal layer. Its average temperature in summer is about −60°F. and in winter −71°F.

INVERSION OF TEMPERATURE. NOCTURNAL STABILITY

When the temperature increases instead of decreases with elevation it is called an inversion of temperature. This occurs on practically all nights, and may be explained as follows: Suppose a quantity of air with a given temperature should rise or be pushed up a short distance on some night. It would expand and cool at the rate of 1.6°F. for 300 feet and find itself cooler than its surroundings. It would then at once drop back again, or rather it would never have risen spontaneously at all. Thus at night the atmosphere is entirely stable and convection is inoperative. This stability of the atmosphere at night is favorable for gas attacks.

HOW THE ATMOSPHERE IS HEATED AND COOLED

Five processes are concerned in heating the atmosphere, namely absorption of insolation, absorption of radiant heat from the earth, conduction from the earth, mixture by winds and convection. There are three processes concerned in the cooling of the atmosphere, radiation to the cool ground and to space, conduction, and mixture by wind. The air radiates heat as well as absorbs it. The ground at night cools rapidly, and the air loses heat by radiation both to the open sky and to the cooling ground. Conduction would cool the 2 or 3 feet of air in immediate contact with the earth's surface, and the wind, though less at night than during the day, would mix this layer with the two or three thousand feet of air above it. Since convection is not operative at night as already explained, the marked cooling must take place very near the earth's surface.

THE AIR TEMPERATURE

The lowest temperature usually comes at about the time of sunrise, and the highest temperature from 2:00 to 4:30 p.m. depending upon the season of the year. The average temperature for the day occurs at about 9:00 a.m. and 8:00 p.m. The rise during the morning and early afternoon is sharp, the curve being convex; the drop during the afternoon and night is long and slow, giving a concave curve. The fact that the highest temperature does not occur at noon when the receipts of energy from the sun are largest, but several hours later, is due to the fact that the earth gives off heat as well as receiving it. The largest amount given off by the earth is at the time it is receiving most insolation. During the early afternoon although receipts of insolation have already commenced to fall off, they are still slightly in excess of expenditures. A rise in temperature will continue until the receipts drop to such a point that expenditures equal the receipts. This will be the time of highest temperature.

DIFFERENCES OF TEMPERATURE WITH ALTITUDE

From 5 to 100 feet above the earth's surface the difference will be comparatively small either during the day or night, probably not more than a degree at most. In the layer of air within five feet of the earth's surface marked differences in temperature will be found. At night the differences in this layer are more marked and will

average as high as 2° to 3°F. with maximum values of 5° or 6°F. The layer of air in immediate contact with the ground is of course the colder. Thermometer shelters should therefore be placed more than 5 feet above the ground. The temperatures observed from 5 to 100 feet will be nearly the same.

TEMPERATURE DIFFERENCES OVER A LIMITED AREA

A small area may be defined as a square mile of surface. During the day, because of wind and convection no appreciable difference of temperature is to be expected in such a limited area. On sunshiny days with little wind, the lower points particularly those in narrow valleys may be a few tenths of a degree warmer than the upper parts of the area. At night the layer of air next to the ground grows cold and denser, and drains like water into valleys or places of low elevation. If wind is insufficient to remove these pockets of cold air, a marked variation will be found over a limited area. The critical wind velocity for most of such areas is about 3 miles per hour. Any higher wind velocity will clear out these cold pockets, but with less wind a variation in temperature appears, and such variations will probably average 4° and may be as much as 10° to 15° at times.

Temperature records are made all over the world, and from this data iso-thermal charts are constructed. The isothermal lines represent the temperature fluctuations over the territory covered by the chart, and may be constructed for months or years.

WIND

Air in motion near the earth's surface and nearly parallel with it, is called wind. All other motions of air are called air currents. The direction, velocity and pressure are to be determined. The wind is named from the direction from which it comes. If from the north to the south it is a north wind. The velocity of the wind and the pressure of the wind are so related that when one has been observed the other can be computed. The pressure in pounds per square foot is equal to a constant by the square of the velocity in miles per hour. The constant varies from 0.004 to 0.007. Using an average value for the constant the relation may be expressed by the formula $P = 0.005V^2$. The following table gives the pressure in pounds per sq. foot for several velocities:

VELOCITY PER HOUR	PRESSURE PER SQUARE FOOT	VELOCITY PER HOUR	PRESSURE PER SQUARE FOOT
miles	*pounds*	*miles*	*pounds*
0	0	40	8.00
5	0.12	50	12.50
10	0.50	60	18.00
15	1.12	70	24.50
20	2.00	80	32.00
25	3.12	100	50.00
30	4.50		

Estimating wind velocities. Wind velocities may be estimated by their effects on common objects.

Miles per hour.	
0	No perceptible movement of anything.
0–4	This just moves the leaves of a tree without moving branches.
4–12	Moves branches of trees and blows up dry leaves or paper from ground.
12–22	Sways branches of trees, blows up dust, and drives leaves and paper rapidly before it.
22–32	Sways whole trees, blows twigs and small branches along the ground, and hinders walking somewhat.
32–72	Breaks small branches, litters ground with twigs and branches, and hinders walking decidedly.
72–on	Causes more or less destruction of everything in its path.

Planetary winds are those that would occur on any planet heated at the equator and turning eastward on its axis. In this way are produced the trade winds and prevailing Westerly winds.

The trade winds blow from the tropical belts of high pressure toward the equator which is an area of low pressure; as a northeast wind in the northern hemisphere and a southeast wind in the southern hemisphere. They blow with a moderate to brisk velocity and especially over the ocean have an unchanged direction for weeks at a time. The velocity increases as they approach the equator. In the northern hemisphere from the tropical belt of pressure poleward, the wind blows from the southwest or some westerly quarter. In the southern hemisphere the wind direction is from the northwest. Both of these winds are called the prevailing Westerlies.

The planetary winds are modified by two factors.

1. The axis of the earth is inclined $23\frac{1}{2}^\circ$ to the plane of the ecliptic which causes a change in the presentation of the northern hemisphere

to the rays of the sun at different parts of its orbit. The modification of the planetary winds produced by this change produces the terrestrial winds. This migration of the sun though 47° as a result of the inclination of the axis causes a migration of the heat belt. As a result the equatorial belt of high temperature and low pressure, and the resultant wind system of the world migrates north and south lagging behind the sun. This migration of the wind system causes three belts known as the subequatorial and two subtropical belts where calms and winds blowing from two opposite directions in the course of a year exist.

2. Continental winds: The earth's surface is not uniform, but part land and part water. The terrestrial winds modified by this diversity of surface are called continental winds. The continents during winter are cooler than the adjoining oceans, while in summer they are warmer. The continents therefore become areas of low pressure in summer and high pressure in winter. This has a tendency to break up the continuous belts. This tendency of the continents to become centers of high pressure in winter with spirally outflowing winds and the reverse in summer is not usually sufficient to cause a complete reversal of wind direction between summer and winter. There are several instances however, where such a complete reversal is brought about, and the winds so produced which change direction with the seasons are called monsoons. A most typical monsoon occurs in India. During the winter a peak of high pressure forms over southern Siberia with a central pressure of 30.50 inches, causing a northeast wind in India, the dry or winter monsoon. During the summer, the equatorial belt of low pressure migrates to the North of India and the wind blowing spirally inward to this belt of low pressure causes wet southwest winds to blow over India. This is the wet monsoon. Other monsoons occur in Spain and Australia.

Land and sea breezes. At the seashore the land is heated more than the water during the day and at night cools rapidly and becomes colder than the ocean. This causes a small convectional circulation known as the land and sea breeze. Thus during the day the land is an area of low pressure and the sea breeze usually begins about 10:00 a.m. and blows with increasing velocity from the ocean to the land. At night the direction is reversed. The land and sea breezes may often be combined with the prevailing wind, in which case they do not blow directly from the sea and the land.

Mountain and valley breezes. Occur in all mountainous regions, but especially in long narrow valleys opening on a large plain below. At night the layer of air next the ground becomes cooled and drains into the valleys. In a long narrow valley this drainage of cold air is felt as a mountain breeze for a few hours after sunset until early morning. During the day the bottom of a valley is heated more than the slopes particularly as one side is nearly always in the shade. The warm air flows off toward the side and is deflected up the sloping sides of the valley causing the valley breeze which flows up the mountain slopes during the day.

THE MOISTURE IN THE ATMOSPHERE

The moisture of the atmosphere, or water vapor is the water present in invisible gaseous form. It varies from almost nothing to 4 per cent as a maximum. Water vapor is lighter than air, the ratio being 100:62. Moist air is therefore lighter than dry air, since a portion of dry air has been replaced by water vapor which is lighter.

Moisture in the air is derived from evaporation. The amount of evaporation depends upon the nature of the surface, the amount of water vapor already in the atmosphere, the temperature, the wind velocity and the barometric pressure. Areas covered with vegetation evaporate one third more than a free water surface under similar conditions. Wind, and high temperature favor evaporation. Evaporation is least when the barometric pressure is largest. The water vapor of the atmosphere is distributed by diffusion, convection and wind.

Capacity of air for water vapor. This depends upon temperature only. With increasing temperature the amount of water vapor that the air can hold increases at an increasing rate. If a given quantity of air contains all the water vapor it can hold it is said to be saturated. Humidity is defined as the state of the atmosphere as regards moisture. It is humidity as well as temperature which makes hot weather uncomfortable. Absolute humidity is the actual quantity of moisture in a given quantity of air, usually expressed as grains per cubic foot or grams per cubic meter. Relative humidity is the ratio of the actual amount of water vapor present to the quantity that would be there if it were saturated. Relative humidity is expressed in per cent. The Dewpoint is the temperature at which air is saturated with moisture so that any further lowering of temperature will

result in precipitation, which may be dew, fog or rain according to circumstances.

Absolute humidity, relative humidity, dewpoint and temperature are so related that, knowing any two of these quantities the other two may be computed from the following table by using the formula $\text{R. H.} = \frac{\text{A. H.}}{\text{Possible amount}}$.

TEMPERATURE	VAPOR PRESSURE	AMOUNT OF WATER VAPOR PER CUBIC FOOT	MASS OF SATURATED AIR PER CUBIC FOOT
°F.	*inches*	*grains*	*grains*
−30	0.010	0.12	650
−20	0.017	0.21	634
−10	0.028	0.35	620
0	0.045	0.54	606
+10	0.071	0.84	593
20	0.110	1.30	580
30	0.166	1.97	568
40	0.246	2.86	556
50	0.360	4.09	544
60	0.517	5.76	533
70	0.732	7.99	521
80	1.022	10.95	509
90	1.408	14.81	497
+100	1.916	19.79	487

TEMPERATURE	VAPOR PRESSURE	AMOUNT OF WATER VAPOR PER CUBIC METER	MASS OF SATURATED AIR PER CUBIC METER
°C.	*mm.*	*grams*	*kgm.*
−30	0.38	0.44	1.45
−20	0.94	1.04	1.40
−10	2.15	2.28	1.35
0	4.57	4.87	1.30
+10	9.14	9.36	1.25
20	17.36	17.15	1.20
30	31.51	30.08	1.15
+40	54.87	50.67	1.11

Thus if the temperature is 70° and the dew point 50°F. find the relative and absolute humidity. From the table we find that air at 50°F can hold 4.09 grains per cubic foot. The absolute humidity is therefore 4.09. Air at 70° can hold 7.99 grains per cubic foot. The relative humidity is therefore $\frac{4.09}{7.99} = 51$ per cent. Or if temperature

is 40°F. and relative humidity is 70 per cent find absolute humidity and dew point. From the table air at 40°F. can hold 2.86 grains per cubic foot. Therefore:

$$70 \text{ per cent} = \frac{\text{A.H.}}{2.86} \text{ and A.H.} = 2.00$$

From the table we find that 2 grains saturates air at a temperature a little above 30°F. The dew point is between 31 and 32°F. Absolute humidity is determined by hygrometers, usually consisting of two U tubes containing $CaCl_2$ and H_2SO_4 through which a known quantity of air is drawn, and the moisture weighed. Relative humidity may be determined more or less accurately by a hair hygrometer. The dew point may be determined by a tin cup and a thermometer. The cup is partly filled with water and ice is added. When the dew point is reached the cup will become coated with moisture.

Effect of moisture on circulation of atmosphere. Moist air is lighter than dry air, and the amount of moisture at the equator is nearly six times the amount of moisture in a similar amount of air at the poles. This therefore operates in the same way as the equatorial temperature to accentuate pressure differences between the equator and the pole, and to cause a general circulation.

Dew. During the day as the temperature rises, moisture is continuously added to the air by evaporation. At night the ground loses its heat rapidly by radiation, and the layer of air close to the earth loses this heat by both conduction and radiation and soon reaches the saturation or dew point. Any further lowering of temperature results in the deposit of dew on the ground. Frost is dew which had formed when the temperature was below 32°F.

Fog. When a layer of air of considerable thickness falls below the temperature of saturation, the moisture is unable to appear as dew at the bottom of the layer but condenses as fog. This condensation largely occurs on dust and other particles in the air. Fog consists of minute droplets of about 0.001 inch diameter with a dust particle for its nucleus. Whenever fog occurs the relative humidity is usually very high, usually at least 90 per cent. On the Atlantic Coast from Maine northwards, fogs occur chiefly during the summer. In New England and the Middle Atlantic States fogs are prevalent during the late winter because warm moisture laden air blows up from the south, and the moisture becomes precipitated as cooling occurs. During late summer when the air is warm and moisture laden the night

temperature may go below the dew point for a considerable thickness of air, and fog forms. Such fogs always commence at night and are thickest at sunrise, and generally disappear during the day. All fogs are caused in one of these two ways, by the transportation of moist air to cooler climates, or by radiation of heat from a moist air.

Clouds. The four types of clouds were originally named cirrus, cumulus, stratus, and nimbus. The cirrus is the delicate, fibrous, hairlike cloud. The cumulus is the lumpy, piled-up cloud. The stratus is the level sheet or layer. The nimbus is the cloud from which precipitation is falling. The international system retains these names with modifications.

Type forms: Cirrus, stratus, cumulus, nimbus.
Intermediate varieties: Cirro-stratus, cirro-cumulus, strato-cumulus, cumulo-nimbus.
Alto forms: Alto-stratus, alto-cumulus.
Fracto forms: Fracto-stratus, fracto-cumulus, fracto-nimbus.
Upper clouds: Cirrus, cirro-stratus 9000 to 46,000 feet height, mean 28,000 feet.
Intermediate clouds: Cirro-cumulus, 7000 to 35,000 feet alto-stratus, alto-cumulus, 2700 to 15,000 feet.
Lower clouds: Strato-cumulus, 1000 to 14,000 feet, 200 to 18,000 feet, average 3600 foot, fracto-nimbus, nimbus.
Clouds formed by daily ascending currents: Cumulus, fracto-cumulus, cumulo-nimbus.
High fogs: Stratus, fracto-stratus 400 to 6000 feet, average 2100 feet.

Sequence of cloud forms. Cloud forms are of little value in forecasting. There is however a fairly regular sequence in passing from a clear sky to stormy weather. First the cirrus, which thickens to cirro-stratus or cirro-cumulus and then becomes alto-stratus or alto-cumulus; next the nimbus appears. In clearing off the nimbus generally breaks up into fracto-nimbus, disclosing an upper cloud area of cirrus or cirro-cumulus. The upper cloud layer gradually disappears, and the lower fracto-nimbus becomes strato-cumulus, fracto-cumulus and finally cumulus.

Clouds move much more rapidly than surface winds. When surface winds are moving with a velocity of 50 miles an hour, cloud velocity may be from 150 to 200 miles per hour.

Cloudiness. The term refers to the amount of sky covered by clouds, and has nothing to do with the kind of clouds. It is usually determined by a naked eye estimation, the sky being divided into

two portions by a small circle at an altitude of 45°. The amount of upper clouds and lower clouds is estimated separately and the estimate is made in fifths since the sum of the two estimates will give the cloudiness in tenths for the whole sky. The sky is called cloudless when no clouds at all are visible; clear when from 0–$\frac{3}{10}$ covered with cloud; partly cloudy for cloudiness between $\frac{3}{10}$ and $\frac{7}{10}$; cloudy when from $\frac{7}{10}$ to all is covered with cloud.

Cloud particles vary in size from $\frac{1}{400}$—$\frac{1}{100}$ of an inch in diameter, and their small size is the reason for their suspension in the air. A water drop $\frac{1}{100}$ inch would ordinarily fall at a rate of less than two inches per second, and thus a very feeble ascending current of air keeps cloud particles in suspension.

Formation of clouds. There are nine methods by which clouds are formed, of which only a few are of major importance.

1. Condensation in warm winds blowing over cold surfaces. A South wind carries warm moisture laden air from Southern countries and carries it north where the colder surfaces cause condensation. These clouds are chiefly of the stratus and nimbus variety and occur from one half mile to two miles above the earth's surface.

2. Condensation in ascending currents. When air rises by convection it expands and cools at a rate of 1.6°F. for 300 feet. When the dew point is reached precipitation occurs. Since convection usually occurs during the day, clouds formed by this method occur only during the day, and are one of the cumulus forms. The height at which a convection cloud should be formed may be computed by dividing the difference between the temperature and the dew point by 1.27 and multiplying by 300. Thus if on a summer afternoon the temperature is 85°F. and the dew point is found to be 70°F., the height of a convection caused cloud would be $\frac{85-70}{1.27}$ 300 or 3543 feet.

3. Condensation in forced ascending currents. Air may be forced to rise by passing over a mountain or in a storm center which is an area of low barometric pressure. Air from such a storm center is one of the chief causes of stratus and nimbus clouds.

Conditions that favor a clear sky. There are two major processes that favor a clear sky. Cold winds blowing over a warm surface and forced descending air currents. Clear skies are very prevalent when the wind is north or northwest, and when there is an area of high barometric pressure which causes descending and outflowing air currents.

Precipitation

Rain. Dew, frost and fog have been considered; in addition rain, snow and hail are included in the forms of precipitation of aqueous vapor. Raindrops are formed from cloud particles. The larger cloud particles fall and coalesce with other particles forming a droplet. As soon as a raindrop begins to fall rapidly it will come into warmer air and a condensation of water vapor will occur on the cold drop. A raindrop thus increases in size due to collision and condensation, until it reaches the base of the cloud and begins its fall to the earth. Raindrops vary in size from very small to $\frac{1}{10}$ of an inch. They may be measured by allowing them to fall into a surface of flour and measuring the size of the dough balls formed.

Snow is simply precipitation while the temperature is below freezing point. Snow crystals are small, flat, and regular six sided crystals with angles of 60° or 120°. If the temperature is below 0°F. fine ice needles are formed instead of flakes. If the temperature in the lower layers of the atmosphere is near freezing, the snow flakes mat together and form large clots. Below freezing they melt. Much winter rain probably left the clouds as snow and melted during the descent.

Hail. There are three kinds of hail. Winter hail is simply frozen raindrops. Soft hail of small white pellets is supposed to be formed from frozen cloud particles mixed with raindrops and compacted by a high wind. It occurs in spring and autumn. During the summer hail never occurs except during a thunderstorm. The hail stones may be very large and consist of concentric layers of compact snow and ice. Such hail is formed in the whirling squall cloud of a thunder shower. The nucleus is carried up into the cloud and coated with snow, it then falls and is coated with water, and is carried up again and frozen, being coated with snow again. This process is repeated until the stone is too heavy to be supported by the whirling wind when it falls.

The amount of precipitation is known as the result of observation for any locality, and can be learned from weather bureau statistics. It may be above 100 inches in the tropics to almost nothing in desert countries.

CYCLONES

Tropical cyclones. Often called typhoons, hurricanes and baguios, are vast atmospheric whirls turning counterclockwise in the northern hemisphere and clockwise in the southern with spirally inflowing winds which usually attain destructive velocities. The pressure is low in the center. The whole formation is from 300 to 600 miles in diameter and moves with a moderate velocity over a fairly well-defined course. Such cyclones occur (1) in the West Indies and Gulf of Mexico, (2) in the China Sea, Philippine Islands and China, (3) on each side of India in the Bay of Bengal and in the Arabian Sea, (4) east of Madagascar, (5), east of Australia near Samoa. They occur mostly during the months of July to October in the Northern hemisphere, and from December to March in the Southern. That is during the hottest months.

Origin of cyclones. Convection is most vigorous in stagnant, overheated, moisture laden air. Suppose that a large mass of quiet, warm, moist air exists. As it rises, it expands, grows cooler, becomes cloudy and may yield rain. The latent heat liberated by the condensation of water vapor lessens the rate of cooling of the rising air and causes a still further rise. Due to the excess of temperature and moisture, the pressure would be less than in surrounding regions. The cooler air forcing its way in, would be deflected to the right in the Northern hemisphere by the earth's rotation, and would approach the center in a counterclockwise spiral. This rotation of air about the center causes centrifugal force which would hold the air away from the center thus causing a still lower barometric pressure. This increased difference in pressure between the center and surrounding regions would drive in the air with greater vigor and cause a further rise of air in the center. This could cause more clouds, more precipitation, more latent heat and a still more vigorous rise. This in turn would make the whirl more vigorous, cause more centrifugal force and a lower barometric pressure in the center. Thus we have a cycle each phase of which tends to increase the whirl. Thus the rapidity of rotation increases as the center is approached, and at the center is a calm area of several miles, with a gently descending air current and no clouds. The outflow of water from a circular wash-bowl with a central vent is exactly analogous.

Extratropical cyclones and anticyclones. An extratropical cyclone is a whirling storm occurring in temperate latitudes. These storms are depicted on the daily weather maps as areas of low pressure.

The ceaseless changes in our weather are due almost entirely to the approach and passage of these areas of low pressure. They are similar to tropical cyclones in many respects but are quite different in other particulars. An extra tropical cyclone is an area of low barometric pressure with spirally inflowing winds turning counter-clockwise in the northern hemisphere and clockwise in the southern. The wind velocity is generally moderate; the accompanying cloud area is immense and precipitation usually falls. The changes in temperature and moisture are large and well marked. The whole formation is from a few hundred to several thousand miles in diameter with an average of 1200 miles and moves with moderate velocity from some westerly to some easterly quarter. There is a marked rise of temperature on the south and east side of the storm where the winds blow from some southerly quarter and a decided drop in temperature on the west side where the winds blow from some northerly quarter. The moisture changes are similar. On the southern and eastern sides of the storm the absolute humidity increases very rapidly. In the central part of the storm both absolute and relative humidity are high. On the west side where the winds are from the north the absolute humidity is low, but the relative humidity remains high because of the small capacity of the air for water vapor. The cirrus clouds are lacking on the west side, but extend far out to the east. The nimbus cloud area which marks the precipitation is not concentric with the isobars but is located chiefly in the southeast quadrant. After studying large numbers of these storms, it is found that they proceed over a very definite course. The weather in any particular locality may be predicted therefore with a high degree of accuracy by noting the direction and rate of approach of one of these storms, and combining this information with local conditions that may cause modifications. Such conditions are: (1) surface topography of the country, such as mountains, nearness to bodies of water, etc., (2) meteorological conditions such as whether the ground is snow covered, conditions of temperature, moisture, etc., (3) the neighboring meteorological formations; that is the position of other lows with their antitheses the highs, with reference to the approaching low in question.

Approach of an extratropical cyclone. The signs of the approach of such a storm are as follows: The wind begins to blow gently from the east, the pressure decreases slightly, cirrus clouds make their appearance, and temperature and moisture begin to increase. Next

the barometer drops a little more, the wind direction changes to the southeast and the velocity becomes a little greater. The cirrus clouds thicken to cirro-stratus or cirro-cumulus and the temperature and moisture continue to rise. In the winter time the weather has begun to moderate. In the summertime it is the beginning of a period of sultriness. The pressure now drops still more, the wind veers a little and blows harder, temperature and moisture are still increasing. Next comes a period of rain or snow with the barometer still dropping until finally reading its lowest. The wind meantime has slackened and is now blowing from the south or southwest. The wind now veers from southwest to west and finally northwest, the barometer begins to rise, the precipitation grows less and temperature and moisture decrease. The nimbus breaks up. The wind continues to blow from the northwest with increasing velocity as the barometer rises and the temperature drops rapidly, and the air becomes dryer. In summer the cool northwest wind has replaced the sultriness of a few days before. In winter the thaw or warm spell has been replaced by a cold snap. This series of weather changes requires from two to four days in winter and nearly double the time in summer for its completion.

Anticyclones. Two valleys cannot occur without a hill between them, and similarly two areas of low pressure cannot exist without a region of high pressure between, these regions are called anticyclones or more simply, highs. A high is an area of high barometric pressure with spirally outflowing winds turning clockwise in the northern hemisphere and opposite in the southern. Wind is moderate; changes in temperature and moisture are large and well marked. The whole formation of about the same size as the extratropical cyclones, moves with moderate velocity from some westerly to some easterly quarter. There is a decided drop in temperature in the northeast portion where winds are from the north and a decided rise in temperature in the southwest and west portions, when the wind is from the south. In winter the highs are larger and the central pressure higher than in summer.

The sequence of changes in the passage of a high is as follows: A low has probably just passed. Its rain area has gone by, and the nimbus has changed to a strato-cumulus. The wind has gone to the northwest, and the temperature has dropped markedly, the air is dryer and the barometer is rising. This indicates that the low is giving place to a high and that several days of good weather may be

expected. So long as the barometric pressure continues to rise, and the wind continues in the northwest it will remain clear and cool, i.e., the high is continuing. When the center of the high is reached the pressure is highest. Then it commences to fall as another low or extratropical cyclone approaches as already described.

In the United States these lows enter mostly from the northwest around Washington and Oregon, a few from the southwest near lower California. Those which enter from the northwest have a tendency to move south in crossing the Mississippi valley, and then recurve to the northeast. Nearly all lows leave by way of the St. Lawrence Valley and pass through the Middle Atlantic States and New England. The average velocity of such storms is 31.7 miles per hour. The winter velocity is nearly twice the summer velocity. The number of lows in winter is almost twice the number in summer.

The thunder shower. A shower is a copious rainfall of short duration. Lightning and thunder are a result of copious condensation in a cloud and play no part in the mechanism of a thunder shower. A thunder shower is an immense cumulonimbus cloud accompanied by copious precipitation, a marked drop in temperature, and a peculiar, outrushing squall wind which just precedes the rainfall. Thunder and lightning are always present and hail sometimes falls. It is a violent local storm and lasts but a short time. The average duration of a thundershower is a little less than two hours. The first thunder is ordinarily heard about an hour before the coming of the rain and the last thunder is heard sometime after the cessation of the rain. The squall wind is a circular whirl on the under surface of the approaching cloud the outrushing of which produces the wind. Thundershowers occur in all parts of the world but are more numerous in the tropics occurring perhaps two hundred days during the year, and diminish in frequency rapidly from the equator to the poles. In the Gulf States there is an average of sixty days in the year with thundershowers, while in New England, the average is about fifteen days. A thundershower covers but a small area, and is a few miles long by a mile or two wide. As it moves across the country it becomes constantly larger and at the end of six or seven hours, which is about the average life of a thundershower, it has a front of 150 to 200 miles long and perhaps 40 miles wide. The average velocity in the United States is between 30 to 40 miles per hour. The majority occur in June, July and August, and during the hottest part of the day, mostly from 3:00 to 5:00 p.m.

A tornado is practically a violent thundershower in which the squall wind is sufficiently large to form a funnel reaching the earth. The velocity of the wind in this funnel may be from 100 to 500 miles per hour, and the barometric pressure in the center is probably reduced to one-half the normal. This sudden lowering of pressure has an explosive effect on buildings in which the air is confined at the original pressure, and the wind completes the damage. The velocity of a tornado is from 20 to 50 miles per hour, so that it passes a given point in less than half a minute. Tornadoes like thunder storms are most frequent from 3:00 to 5:00 in the afternoon.

WEATHER PREDICTIONS

Weather is the condition of the atmosphere at any particular time and place. It is determined by the six weather elements, temperature, pressure, wind, moisture, cloud and precipitation. To predict the weather is to foretell the values that these different weather elements may have at some future time. The weather is the composite or resultant of three factors as follows: (1) The typical or normal condition of the atmosphere which would exist if there were no disturbances or local influences. (2) The disturbances caused by such passing atmospheric conditions as extratropical cyclones, anticyclones, and thundershowers. (3) Local influences such as land and sea breeze, mountains or the presence of large bodies of water. The typical or normal weather may be known as the average of a large number of observations for that place and season, and these normal values when once determined hold good for all time. Thus normal weather is known for weeks or years ahead. The local influences are usually unimportant and can be estimated with fair precision. The chief difficulty is therefore in determining the influence of the passing meteorological formation. If all lows and highs were typical, weather forecasting would be easy and most accurate. Unfortunately the lows and highs are so erratic as regards path, velocity and other characteristics that forecasting is not always easy. In forecasting, therefore, the first thing is to estimate the influence which the passing meteorological formation will exert during the next day or two. The local influences must next be estimated. When these two have been combined with normal weather, the resultant is the predicted weather.

The United States Bureau receives meteorological observations at 8:00 a.m. and 8:00 p.m. from the entire United States. Based on

these observations, a weather map and forecasts are issued. The predictions based on the 8:00 a.m. observations are for 36 hours, while those based on the 8:00 p.m. observations are for the following forty-eight hours. The forecast predicts temperature, wind, amount of cloud and kind and quantity of precipitation. Pressure and moisture are never predicted. Cold waves, frosts and high wind velocities are also predicted. All forecasts and warnings of high winds, cold waves and frosts come from the forecast centers.

Locating the storm center twenty-four hours ahead. Whether a low or high is expected the first thing is to determine where its center will be twenty-four or forty-eight hours ahead. This can be determined with fair accuracy for twenty-four hours but forty-eight hours ahead can only be roughly estimated. The general rule is to determine the track which the low seems to be following, and then put it ahead on this track the distance normally covered in twenty-four hours. The normal velocity is about 900 miles a day in winter and about 600 in summer. Long experience is necessary before reliable forecasts can be made. Certain rules are of assistance in this connection.

1. Lows near each other tend to coalesce and become more intense than either component.
2. Lows and highs repel each other.
3. Lows tend to follow the record of previous days as to speed of movement.
4. Lows tend to move toward the areas of greatest rainfall during the preceding twenty-four hours.
5. Lows tend to move toward the areas of highest dew point.
6. Lows tend to move toward areas of least wind velocity.
7. Lows tend to grow more intense as they approach bodies of water such as the Great Lakes or the Atlantic Coast.
8. Lows tend to move faster as the pressure grows less.
9. Lows with winds above normal weaken, while lows with wind below normal tend to increase.
10. Lows on a curved track tend to move faster when they turn northeastward.

Prediction of frost. This is predicted just like any other temperature. On the basis of the weather map the forecaster must estimate the probable minimum temperature on the following morning. He will be guided largely by the probable clearness of the sky during the night and the probable wind velocity. A clear sky and absence of wind are essential for a large drop in temperature and a consequent frost. The temperatures taken are in thermometer shelters. Three

things must be taken into account. (1) Plant temperatures go below the real air temperature because they are not sheltered and are free to radiate heat. (2) Vegetation is located near the ground instead of the height of a thermometer shelter. (3) The variation in temperature over a limited area is often considerable. Thus the temperature of vegetation may be from 5° to as much as 20° lower than the estimated minimum of the thermometer shelter.

Accuracy attained in predictions. This is between 80 and 85 per cent. The accuracy that can be attained in different parts of the country is very different. California is perhaps the easiest and New England the hardest to predict. The accuracy of forecasts of cold waves is only about 70 per cent. An accuracy of 85 per cent means that on the average, one day in seven will see a complete failure. However a guess should yield an accuracy of but 50 per cent. Weather forecasting is like medicine in that it can never be an exact science like astronomy. The weather forecasts are nevertheless extremely useful, and save in lives and property from three to ten times their cost.

Forecasts from local observations. A good idea of the coming weather can be formed in this way although exact and definite prediction requires the use of the daily weather map. The following is printed on the face of all maps of the United States Weather Bureau:

When the wind sets in from points between south and southeast and the barometer falls steadily, a storm is approaching from the west or northwest, and its center will pass near or to the north of the observer within twelve to twenty-four hours, with winds shifting to northwest by way of southwest and west. When the wind sets in from points between east and northeast, and the barometer falls steadily, a storm is approaching from the south or southwest, and its center will pass near or to the south or east of the observer within twelve to twenty-four hours, with winds shifting to northwest by way of north. The rapidity of the storms approach and its intensity will be indicated by the rate and amount of the fall in the barometer.

REFERENCE

Willis I. Milham: Meteorology.
The Macmillan Company, New York, 1918.

CHAPTER V

Characteristics of Smoke and Gas Clouds

A smoke is merely a cloud of minute particles of some solid suspended in the air. Mists or fogs are very similar but consist of minute droplets of liquid suspended in the air. The particles of a smoke or fog vary in size from those which are just large enough to be resolved by the unaided eye to those which approach the size of single molecules. Thus smoke particles are about the same size as the suspended particles in a colloidal solution, and a smoke may be regarded as a colloidal suspension in which the medium is a gas (air) instead of a liquid. As one of the main objects of a toxic smoke is to penetrate the gas mask used by the enemy, a constant endeavor is being made to generate smoke clouds having as great a number as possible of the smallest possible particles.

DIFFUSION OF SMOKE PARTICLES

Smoke particles exhibit the Brownian movements, which are probably due to the collisions of the molecules of the air with the particles of the smoke. As a result of this movement the smoke particles can gradually diffuse and spread. The larger particles of the smoke would diffuse more slowly than the smaller ones, not only because of their greater mass and inertia, but because of the greater resistance of the air. Winds and convectional currents are far more important in the dispersion of smokes than is diffusion; in fact diffusion is negligible as compared with wind.

SETTLING OF SMOKE PARTICLES

Smoke particles settle out of the air very slowly at a rate depending upon their size, the larger settling out more rapidly than the smaller. The following table gives the estimated rates of settling in air for spherical particles of unit density and various sizes.

DIAMETER OF PARTICLES	RATES OF SETTLING
10^{-2} cm.	30 cm./sec. 1080 m. per hour
10^{-3} cm.	0.3 cm./sec. 10.8 m. per hour
10^{-4} cm.	3×10^{-3} cm./sec. 10.8 cm. per hour
10^{-5} cm.	3×10^{-5} cm./sec. 1.08 mm. per hour

Thus a particle having a diameter of 10^{-4}, which is the most common size of smoke particles, would fall about 0.071 inch per minute, a rate so slow as to be negligible for practical purposes.

GROWTH OF SMOKE PARTICLES

Like particles in colloidal solution particles of a smoke have a tendency to increase in size for several reasons. Cohesion and coalescence of the particles may occur as the particles are brought in contact with each other by the Brownian movement or air currents. This agglomeration of particles is not marked when they are dry, but is more pronounced with liquid particles, or particles of a deliquescent solid which have condensed some water upon their surfaces. The growth of particles by agglomeration is much more rapid in a dense cloud than in a thin one, since the particles are nearer and will come into contact with each other more readily.

Finally, the smaller particles vaporize relatively more rapidly than the larger particles because they have a greater surface in proportion to their weight. The surface of a body varies as the square of its diameter, while mass varies as the cube of the diameter. Thus doubling the diameter of a spherical body increases the surface fourfold but the weight eightfold.

Thus for several reasons the larger particles in a smoke cloud increase in number and size at the expense of the smaller particles. The increase in size increases the rate of settling, and eventually this leads to a material decrease in concentration of a cloud even when it is not diluted by air currents. The most stable smoke will be one whose particles are very small and consist of a dry non-volatile and non-deliquescent solid.

CLASSES OF SMOKES

Those used in warfare are (1) screening smokes which interfere with visibility, and (2) toxic smokes, which are poisonous or irritant. The toxic smokes are intended to penetrate a mask, and will be discussed further elsewhere.

A screening smoke attains its object partly by obstructing the rays of light, but still more by their scattering due to the reflection of the rays from the individual particles of the smoke. This scattering is dependent primarily upon the concentration of these particles or the number in a given volume. A screening smoke is therefore more

effective if its particles are very finely divided, as this increases the number of particles in a given volume and increases light reflection. The value of a screening smoke is dependent on its power to obscure objects behind it, and the relative value of screening smokes is determined by their "total obscuring power" (abbreviated T.O.P.). The T.O.P. of a smoke is defined as the area in square feet which may be covered by the smoke from one pound of smoke producing material, spread out in a layer of such thickness and density that it will just obscure the filament of a 40 watt Mazda lamp. Density as used in this connection refers to the effect of the smoke upon light, and for a particular smoke material having particles of a given size, the density of the smoke will be proportional to the concentration of the smoke material in the cloud.

In actual practice the density of a smoke is expressed as the reciprocal of the depth in feet of the smoke layer which will just obscure the filament of a 40 watt Mazda lamp. A cloud of unit density is one of which a one foot layer will obscure the lamp. If the density of a cloud is $\frac{1}{2}$ it will require two feet of it to obscure the lamp. One pound of phosphorus can produce 4600 cubic feet of smoke of a unit density. Hence the T.O.P. of phosphorus is 4600.

THE BEHAVIOR OF A SMOKE CLOUD

It has been shown that the diffusion and settling of smoke particles has little bearing on the travel and spread of a smoke cloud. Even in very dense smokes, the weight of the smoke material in a cloud is only a small fraction of one per cent of the weight of the air in the same cloud. A smoke cloud can therefore be regarded as merely so much air which is distinguished from the general atmosphere only by the fact that a small amount of foreign material is suspended in it. The movement of the cloud will therefore be merely the movement of the air. This explains the characteristics of the movements of clouds. Thus the rising of the smoke cloud is due to upward currents of air. If the cloud is released in warm air it will rise, as the warm air expands, becomes lighter than the surrounding air and rises. If released in cold air or air where these convectional currents are absent, the smoke will cling to the ground.

ANGLE OF SPREAD

If we have a large volume of smoke, emitted from a single small source, and the smoke is carried away from its source by a steady wind, the smoke will spread out in the form of a cone shaped cloud

whose apex is the source. Such a cloud will continue to spread at a uniform rate for a considerable distance. The rate of spread varies with the velocity of the wind, but with velocities of 10 to 12 miles per hour which are most suitable for the use of Chemical Warfare material, the angle of spread is from 10° to 15° on each side of the axis of the wind. The spread of a cloud occurs because even in a steady wind, the air does not move in straight lines but is full of whirls and eddies which carry wisps of the smoke out of the main body of the cloud. In a steady wind these eddies will be fairly uniformly distributed, and the smoke will continue to spread at a fairly uniform rate, and the boundaries of the smoke cloud will be approximately straight lines radiating from the source of the smoke.

But after the smoke has reached some distance from its source, the angle of spread appears to become smaller and the boundary lines of the smoke gradually approach a direction nearly parallel to the direction of the wind. This is due to the fact that at a considerable distance from its source the smoke has become diluted on the edge of the cloud by admixture of air from the outside to such an extent that it becomes less visible, and finally the edge of the cloud becomes invisible. Hence the decrease in the angle of spread at a distance from the source is probably only apparent, at least along the sides of the cloud near the ground.

But at the top of the cloud there is a real decrease in the angle of spread. The wind velocity is always greater at a distance above the ground than it is near the surface. For this reason the upper part of the cloud travels forward faster than the lower part, and this factor becomes operative as soon as the angle of spread takes the upper portion of the cloud sufficiently high to encounter higher wind velocities. This flattening effect becomes more pronounced as the cloud proceeds, hence the height to which an appreciable cloud will rise is limited, and the height to which it will rise depends upon the difference in velocity of wind at the surface and higher. The smaller this difference the higher the visible cloud will rise. Because of this flattening of the cloud the outer air will have a better opportunity to enter the cloud from above and dilute it as it moves forward. Also the front of the cloud will not present a vertical wall of smoke but the upper part of the cloud will proceed some distance in advance of the lower part near the ground. It appears as though the cloud dragged along the surface of the ground. This effect is also more pronounced the greater the difference in the two wind velocities, and the greater the distance of the cloud from its source.

Finally from this projecting or overhanging front of the cloud, there will be more or less smoke carried downward by vertical eddies, and this produces the effect of a rolling or tumbling front. The forward and upper part of the cloud, already considerably diluted by air entering from above or at the sides, is further diluted by air from the front of the cloud. Therefore, a person standing in the path of an approaching cloud encounters, not a sudden high concentration of smoke or gas, but first an almost imperceptible trace of the cloud and gradually as the cloud advanced he will encounter higher and higher concentrations until the maximum is reached. And the farther the cloud has travelled from its origin, the more gradual will be the rise in concentration of the smoke material from the front of the cloud toward its interior.

EFFECTS OF TERRAIN AND WEATHER

The direction and intensity of the wind near the earth's surface may be materially modified by the relative positions, prominence and direction of hills, and valleys, and by obstacles to air movements, such as high trees. On a clear day in summer, the sun heats the earth and the lower stratum of air and thus convectional currents are constant; while on a clear night with a high barometer there may be even descending air currents. All atmospheric factors that affect movements of air affect the movement of clouds. Moreover the temperature will have considerable influence upon all clouds depending upon or affected by atmospheric humidity, which is much higher in warm than in cold weather.

The above general principles regarding the behavior of smoke clouds apply equally well to gas clouds. However, in the case of gas clouds it must be remembered that most warfare gases are much heavier than air, and therefore, drain off into all valleys and depressions in the ground. Tall grass, bushes, trees, buildings, etc., retard the movements of air and gas clouds making them more persistent. In dugouts, cellars, buildings, shell holes, deep hollows, and woods, gases persist longer than in the open. In light winds the contour of the terrain is the controlling factor of wind direction. A valley whose main direction deviates up to 45° from the direction of the wind will usually control the course of the air currents and of the gas clouds.

It is inexpedient to use gas in winds of considerable velocity since under such conditions the gas cloud is quickly dispersed and pro-

duces no casualties. Under such conditions gas attacks by cloud need not be feared. Surprise shells are effective in winds that blow not over 10 miles an hour in the open, or not over 25 miles an hour in thick woods.

HUMIDITY AND TEMPERATURE

Humidity and temperature effect gases differently. A light rainfall has little effect on most gases. Even a heavy rain effects mustard very little. On the other hand phosgene or lewisite would be largely hydrolyzed and become relatively ineffective in wet weather. Foggy weather is distinctly advantageous for the use of gas clouds if the gases are not destroyed by moisture. Warm sunny days make the use of most gases ineffective, especially in clouds, as convectional currents are strong. On the other hand very persistent substances like mustard, which have a high boiling point, are diffused more readily on hot days. In the winter the volatilization of mustard is very slow.

CHAPTER VI

Classification of Chemical Warfare Materials

The term gas warfare is not literally correct and is no longer used, because many chemical warfare agents are liquids or solids at the ordinary temperatures encountered in the field. Thus we may classify chemical warfare materials according to their physical state, as gases, liquids and solids. However, it must be remembered that although liquids like mustard may act as such, they also enter the gaseous state at certain temperatures, and as a gas may thereby become more widely disseminated and produce many more casualties than would be possible by a liquid which is necessarily confined to a small area. Solids are only toxic when they are enabled to become dissolved in some manner in the body fluids. Accordingly solids are generally vaporized by explosive shell, by heat or some other method, whereby they are dispersed through the atmosphere as finely divided particles or smokes, that may be inhaled. Thus we see that although the term gas warfare is not technically correct, it does convey a very definite and accurate idea of the method by which chemical warfare agents are made effective.

Depending upon physical condition and physical constants, particularly the vapor pressure, chemical warfare materials are also classified as persistent and non-persistent. Gases in general are non-persistent, while liquids and solids are apt to be persistent. Thus chlorine and phosgene are non-persistent. Chlorpicrin is more persistent than chlorine or phosgene because its boiling point is higher and it is a liquid at ordinary temperatures. Mustard has a still higher boiling point and is quite persistent, while solid chloracetophenone will persist for weeks or months. Solids that have become vaporized as smokes behave like gases and are relatively non-persistent.

The tactical use of chemical warfare materials is largely governed by this property of persistency. Thus chemical agents are used:

1. To produce casualties. Non-persistent agents of the phosgene type not only produce casualties, but are pronounced lethal agents, while persistent agents like mustard, seldom cause death but do produce many more casualites than the non-persistent agents.

2. To harass and lower morale. Both persistent and non-persistent types are used for this purpose according to circumstances.

3. To render strong positions untenable. From the nature of the case only persistent agents can be used for this purpose.

4. To conceal movements and lead the enemy to draw wrong conclusions. Smokes are chiefly used because of their obscuring power. Relatively non-persistent.

5. To damage enemy material and supplies. For this purpose incendiary materials and corrosive agents are chiefly used. Chlorine and phosgene both cause corrosion of metals and may put rifles and machine guns out of commission temporarily. Mustard and arsenical agents may render food unfit for consumption.

PHYSIOLOGIC CLASSIFICATION OF CHEMICAL WARFARE AGENTS

The most useful classification for medical purposes is according to physiologic action. This action is very complex in most cases, and different parts and organs of the body are usually simultaneously affected. Nevertheless the action of the more important agents is especially effective upon some particular part of the body, and they may therefore be classified in accordance with their most important physiologic effects as follows:

1. Pulmonary irritants. The main characteristic of these is that they cause irritation and damage of the respiratory passages, and especially of the pulmonary alveoli with the production of acute pulmonary edema and resulting anoxemia and death by asphyxia. In general these gases are also quite effective lachrymators although far less powerful in this respect than the agents classed as lachrymators. Examples: Chlorine, phosgene, chlorpicrin.

2. Vesicants. These are so classed because they cause inflammation and blistering of the skin. They also cause acute conjunctivitis and intense inflammation of the respiratory passages. They do not cause pulmonary edema. Examples: Mustard, lewisite.

3. Lachrymators. Extremely low concentrations exert an intense irritant action on the eyes, and cause so profuse a flow of tears and so much pain that vision becomes impossible. These effects are temporary. They are not lethal. Examples: Chloracetophenone, brombenzylcyanide.

4. Irritant gases. These were formerly called sternutators and toxic smokes. They cause sneezing and irritation of the eyes, nose

SUBSTANCE	CHEMICAL FORMULA	DATE OF INTRODUCTION	BOILING POINT	VAPOUR PRESSURE IN MM. MERCURY			APPROXIMATE CONCENTRATION TO INCAPACITATE A MAN IN A FEW SECONDS OWING TO SENSORY IRRITATION OF THE EYES (LACHRYMATION) OR UPPER RESPIRATORY PASSAGES (PAIN, COUGHING)		APPROXIMATE CONCENTRATION WHICH IF BREATHED FOR MORE THAN ONE OR TWO MINUTES WOULD CAUSE DEFINITE BRONCHIAL OR PULMONARY LESIONS			BY WHOM USED
				At 10°C.	At 20°C.	At 30°C.						
Acute lung irritants												
			°C.	mm.	mm.	mm.		mgm.			mgm.	
Chlorine	Cl_2	1915	− 33.6	3,760	5,030	6,650	1:10,000	= 0.29	1:10,000	=	0.29	B., F., G.*
Phosgene	$COCl_2$	1915	+ 8	836	1,215	1,750	1:100,000	= 0.04	1:50,000	=	0.08	B., F., G.
Chlormethyl-chloroformate ("Palite," French)	$Cl{\cdot}COO{\cdot}CH_2{\cdot}Cl$	1915	+105	3.6	5.6		1:100,000	= 0.05	1:10,000	=	0.5	F., G.
Trichlormethylchloroformate ("Diphosgene," British "Surpalite," French)	$Cl{\cdot}COO{\cdot}CCl_3$	1916	+128	5	10.3	16.3	1:200,000	= 0.04	1:50,000	=	0.162	F., G.
Chloropicrin	$CCl_3{\cdot}NO_2$	1916	+112	10.8	18.9	30.5	1:200,000	= 0.026	1:50,000 (cumulative)	=	0.1	B., F., G.
Stannic chloride	$SnCl_4$	1916	+114	10.3	18.6	31.3						
Phenyl-carbylamine-chloride	$C_6H_5N{:}CCl_2$	1917	+209						1:10,000	=	0.8	B.
Cyanogen bromide	CNBr	1918	+ 61.3 (M.P. 52°)	63.3 (15°), 119.5 (25°)			1:200,000	= 0.035	1:10,000	=	0.71	G.
									1:30,000	=	0.14	A.
Dichlor-methyl-ether	$(CH_2{\cdot}Cl)_2{:}O$	1918	+105				1:10,000	= 0.47	1:10,000	=	0.47	G.
Lachrymators												
Benzyl bromide	$C_6H_5CH_2{\cdot}Br$	1915	+198	about 1 mm. at 25°			1:2,000,000	= 0.00349				G.
Xylyl bromide	$CH_3C_6H_4{\cdot}CH_2{\cdot}Br$	1915	+216 to 220				1:2,000,000	= 0.00378				G.
Ethyl-iodoacetate	$CH_2I{\cdot}COOC_2H_5$	1916	+180	0.28	0.54	0.87	1:2,000,000	= 0.00437	1:50,000	=	0.17	B.
Bromacetone	$CH_3{\cdot}CO{\cdot}CH_2Br$	1916	>+136	1			1:2,000,000	= 0.0028	1:10,000	=	0.56	F., A.
Monobrom-methylethyl-ketone	$CH_3Br{\cdot}CO{\cdot}CH_2{\cdot}CH_3$ or $CH_3{\cdot}CO{\cdot}CHBr{\cdot}CH_3$	1916	+145 +133				1:500,000	= 0.0126	1:5,000	=	1.26	G., A.

Dibrom-methyl-ethyl-ketone	$CH_3 \cdot CO \cdot CHBr \cdot CH_2Br$	1916	>+ 53 (decomp.)				1:500,000 = 0.0188	1:5,000 = 1.88	G., A.
Acrolein	$CH_2 \cdot CH \cdot CHO$	1916	+ 52	140					F.
Methyl-chlorsulphonate	$SO_2 < {OCH_3 \atop Cl}$	1915	+132						G.
Paralysants									
Hydrocyanic acid	HCN	1916	+ 26.5	(455 at 15°)	603	860	1:2,000 = 0.55 (immediately fatal)	Practically no cumulative action	B., F.
Sulphuretted hydrogen	H_2S	1916	− 61.8	16,500			1:1,000 = 1.39 (immediarely fatal)	1:10,000 = 0.139 (affects eyes and lungs)	B.
Sensory irritants of eyes, nose and chest (sternutators)									
Diphenyl-chlorarsine	$(C_6H_5)_2AsCl$	1917	+333 (M.P.+43°)	0.02 mm. at 50°			1:10,000,000 = 0.001	1:50,000 = 2.16	G.
Diphenyl-cyanarsine	$(C_6H_5)_2AsCN$	1918	>+300 (M.P.+31°)				1:10 000,000 = 0 001	1:50,000 = 2.08	G.
Ethyl-dichlor-arsine	$C_2H_5AsCl_2$	1918	+156				1:500.000 = 0 014	1:20.000 = 0.35	G.
Ethyl-dibrom-arsine	$C_2H_5AsBr_2$	1918							G.
N. ethyl carbazol	$(C_6H_4)_2N \cdot C_2H_5$	1918	>+190 (M.P.+68°						G.
									G.
Vesicants									
Dichlor-ethyl-sulphide (Thio-diglycol-chloride: "Mustard Gas," British; "Yperite," French)	$(CH_2Cl \cdot CH_2)_2S$	1917	+217 (M.P.+13°)	0.01	0.06	0.09		1:1,000,000 = 0.0065 with 60 minutes exposure	B., F., G., A.

* B. = British; F. = French; G. = Germans; A. = Austrians.

Fillings of German artillery and trench mortar gas shell

CODE NAME OR DISTINCTIVE MARKING ON THE SHELL		CHEMICAL SUBSTANCE	USED FOR
German	English equivalent		
T-Stoff	T-substance	Xylyl bromide and/or benzyl bromide	Artillery
T-grün	Green T-substance	Xylyl bromide + brommethyl-ethyl ketones	Artillery
K-Stoff	K-substance	Chlormethyl-chloro-formate	Artillery and trench mortars
C-Stoff (K2-Stoff).	C-substance (K2-substance)	Trichlormethyl-chloroformate	Artillery and trench mortars
B-Stoff	B-substance	Brom-methyl-ethyl-ketones	Trench mortars
D-Stoff Grünkreuz	D-substance green cross	Phosgene	Trench mortars
		Trichlormethyl-chloroformate; or brom-methyl-ethyl-ketones; or phenyl-carbylamine chloride	Artillery
	H. E. green cross (Shell marked with a double or Lorraine cross in green)	Phosgene + large H.E. charge	Artillery
Grünkreuz 1	Green cross 1	Trichlormethyl-chloroformate + chloropicrin	Artillery
Grünkreuz 2	Green cross 2	Phosgene + trichlor-methyl-chloroformate + diphenyl-chlorarsine	Artillery
Grünkreuz 3 (vide infra Gelbkreuz 1)	Green cross 3 (vide infra, Yellow cross 1)		

Blaukreuz	Blue cross	Diphenyl-chlorarsine; or ditto + diphenyl-cyan-arsine or N. ethyl carbazol. Large H.E. charge in all cases	Artillery
Gelbkreuz	Yellow cross	Dichlor–ethyl–sulphide + 15–20 per cent of solvents, e.g., carbon tetrachloride, chloro-benzene, nitro-benzene	Artillery and trench mortars
	H.E. yellow cross (shell marked with double or Lorraine cross in yellow)	Dichlor-ethyl-sulphide and solvents + large H.E. charge	Artillery
Gelbkreuz 1 (re-named Grünkreuz 3, July 1918	Yellow cross 1 re-named green cross 3 July 1918)	Ethyl dichlor-arsine + dichlor-methyl ether or ditto + ethyl-dibrom arsine	Artillery

Fillings of Austrian artillery gas shell

CODE NAME OR DISTINCTIVE MARKING ON THE SHELL		CHEMICAL SUBSTANCE
German	English equivalent	
C-Stoff	C-substance	Cyanogen bromide
B-Stoff	B-substance	Bromacetone
	CB-shell	Cyanogen bromide + bromacetone; or brom-methyl-ethyl-ketones; or cyanogen bromide + brom-methyl-ethyl-ketones

Fillings of German gas projector bombs

Phosgene
Phosgene + trichlor-methyl-chloroformate + chloropicrin
Phosgene + chloropicrin
A mixture of about equal parts of diphenyl-chlorarsine and H.E.

Filling of German gas hand grenades

TYPE OF GRENADE	DISTINGUISHING MARK	CHEMICAL SUBSTANCE
Spherical grenade	Red "B"	Brom-methyl-ethyl-ketones
Spherical grenade	Red "C"	Methyl-chlorsulphonate + 5 per cent of methyl sulphate
Stick grenade	Blue "C"	Diphenyl-chlorarsine + an equal weight of H.E.

Note: Spherical glass hand grenades filled with chlorsulphonic acid were also employed for smoke-producing purposes.

PERIOD	METHOD OF EMPLOYING GAS	GAS USED	INDIVIDUAL PROTECTION FOR TROOPS
1. April and May, 1915 (6 attacks)	Cylinders (i.e., cloud gas)	Chlorine	Nil at first; cotton waste respirators in last attack
2. April, 1915, to July, 1916	Gas shell	Lachrymators, e.g., xylyl bromide	Nil at first, then in succession—cotton waste respirators, goggles, fabric helmets (Hypo.P,PH, and PHG), a few large box respirators for special units
3. December, 1915, to August, 1916 (6 attacks)	Cylinders	Chlorine and phosgene	Fabric helmets (P in December, subsequently PH or PHG); a few large box respirators for special units
4. July, 1916, to July, 1917 ("lethal" shell period)	Gas shell	Phosgene, diphosgene, chloropicrin, also lachrymators	PH or PHG helmets at first. Small box respirators introduced end of August, 1916, issue complete early in 1917
5. July, 1917, to end of the war (mustard gas period)	Gas shell	Yellow cross (mustard gas; green cross (phosgene, diphosgene, chloropicrin, + in some cases chlorarsines); blue cross (chlorarsines + large H.E. charge)	Small box respirators (from April, 1917, onwards these contained a filter to remove solid substances existing in the particulate state in gas clouds)
6. December, 1917, to May, 1918 (16 bombardments)	Projectors	Phosgene, diphosgene, chloropicrin, chlorarsines	Small box respirators

and throat even in low concentrations, nausea and certain nervous phenomena. They cause little permanent damage and are practically never lethal.

5. Direct poisons of the nervous system. In sufficient concentrations, these act with great rapidity, causing a cessation and finally total abolition of the functions of the central nervous system. Although such agents are highly lethal they produce few casualties. Example: Hydrocyanic acid.

6. Gases which act by interfering with the respiratory properties of the blood. These may act in several ways. Thus arseniuretted hydrogen causes destruction of the red corpuscles, accompanied by hemoglobinuria and jaundice; some gases cause the alteration of hemoglobin into methemoglobin; carbon monoxide forms a firm combination with hemoglobin thus preventing the hemoglobin from combining with oxygen.

This last group is relatively unimportant in chemical warfare, as they have never found practical application as war gases. This classification will be followed in describing the physical and chemical characteristics of the various agents, the symptoms and pathological changes produced by them, and the methods of treating the various types of gas poisoning.

The preceding tables from the British "History of the Great War," 1923, Volume II, give the different chemical warfare materials used during the war, with the manner in which they were used, and the approximate dates of introduction.

CHAPTER VII

Pulmonary Irritants—Chlorine, Phosgene, Chlorpicrin, etc.

CHLORINE

Chlorine is of historical interest because it was the first material used by the Germans in Chemical Warfare. Although it seems that chlorine would not be used alone in future warfare owing to the fact that other gases are much more efficient, it is manufactured constantly in considerable quantity for the ordinary chemical industries of peace, and could therefore be more readily produced in quantity for war usage by nations that might not be prepared to produce the more complicated toxic gases. It is also an essential ingredient of almost all chemical warfare material.

Chlorine is a heavy, yellowish green gas. Its atomic weight is 35.79. A liter of chlorine under standard conditions weighs 3.22 grams while a liter of air weighs 1.293 grams. Hence the vapor density of chlorine as compared with air is 2.49, and chlorine is approximately 2.5 times as heavy as air. Chlorine therefore clings to the ground and flows into depressions. At 15°C., it requires only from 4 to 5 atmospheres pressure to liquefy it. One liter of liquid chlorine at 25°C. will produce 434 liters of chlorine gas.

Chlorine is one of the most active elements known and for this reason is never found free in nature. The natural compounds of chlorine are all chlorides of metals, chiefly sodium and potassium chloride. Common salt is the source of practically all chlorine and chlorine compounds found on the market. Chlorine is best made by the electrolytic dissociation of a saturated and purified solution of common salt. The great chemical activity of chlorine is manifested chiefly only in the presence of moisture, which apparently acts as a catalyst. When chlorine is dehydrated as completely as possible it has little or no action on even such active elements as phosphorous, sodium, iron, etc. As a result of this peculiar action of chlorine, the thoroughly dried liquid gas can be stored in plain steel cylinders. Chlorine cylinders are sometimes received in a badly corroded condition. This is caused by slight leakage from the valve while in transit. The escaping chlorine combines at once with the metal with the aid

of the moisture which is always present in the atmosphere. Chlorine is used by the medical department for the sterilization of water and the preparation of Dakin's solution, and it is important to understand that this corrosion when it occurs, is not caused by impurities in the chlorine.

PHOSGENE

Phosgene ($COCl_2$) is carbonyl chloride or carbon oxychloride. It was called phosgene because it was first formed from carbon monoxide and chlorine under the influence of light (*phos* light and *gene* generating). Phosgene boils at 8.2°C. and freezes at −75°C., and it is therefore a gas at ordinary temperatures unless it is under pressure. It is colorless, has a rather characteristic odor described as the odor of musty hay, green corn or green apples. It hydrolyzes readily into hydrochloric acid. The molecular weight of phosgene is 99 and that of air is 28.95. The density of gaseous phosgene as compared with air is therefore 3.505. Owing to this high vapor density phosgene will hug the ground, flowing into ravines, trenches and dugouts unless it is carried upward by convection currents. Since phosgene is a gas under ordinary conditions it is practically non-persistent, and will disappear from any locality as fast as the wind moves through that place.

Phosgene has a high vapor pressure, ranging from 335 mm. Hg at −13.7°C. to 760 mm. Hg at 8.2°C. its boiling point. The higher the vapor tension of a gas, the greater is the amount of that gas that can be contained in a given amount of air. The amount of phosgene vapor present in one liter of air saturated with the gas would be 2.1 grams at −13.7°C., or 2100 mgm. per liter. Since as little as 1 mgm. per liter may be lethal if the exposure lasts more than a few minutes, and is quite capable of producing casualties, it can be readily seen that even in cold weather and a light wind, effective concentrations of phosgene may be secured as a result of the great toxicity of phosgene and the wide margin between the theoretically possible concentration and the lethal dose. It is the most effective of the pulmonary irritants. Were it not for the fact that the mask affords complete protection no army could stand against it. To the end of the war its use continued to produce many casualities when gas discipline was relaxed.

Chemical properties. Phosgene is a fairly stable compound at ordinary temperatures and in the absence of moisture. In contact with water phosgene is rapidly hydrolyzed, especially at elevated temperature, forming carbon dioxide and hydrochloric acid, according to the following equation:

$$COCl_2 + H_2O = 2HCl + CO_2$$

this is the most important property of phosgene. There is always more or less water in the air and the extent of the hydrolysis that will occur is proportional to the humidity of the atmosphere. It follows, therefore, that phosgene cannot be successfully used in misty or rainy weather. Because of this formation of hydrochloric acid, iron and steel articles will be corroded by phosgene, and clothing may be injured. The toxic action of phosgene is also due to this liberation of hydrochloric acid in the tissue of the lungs.

Bases react with phosgene, forming a chloride and a carbonate as follows:

$$COCl_2 + 4NaOH = 2NaCl + Na_2CO_3 + 2H_2O$$

Calcium oxide reacts similarly. "Soda-lime," a mixture of sodium hydroxide and calcium hydroxide is used in the gas mask canister as an absorbent of phosgene. Ammonia reacts with phosgene independently, forming urea and hydrochloric acid, thus:

$$COCl_2 + 2NH_3 = CO(NH_2)_2 + 2HCl$$

Phenol (C_6H_5OH) reacts with phosgene in two different ways depending upon the relative amounts of the two reacting substances. With one molecule of phenol one molecule of phosgene forms phenyl chloroformate, $ClCO{\cdot}O(C_6H_5)$ a derivative of formic acid, $HCO{\cdot}OH$. With two molecules of phenol, phosgene forms phenyl carbonate $CO(OC_6H_5)_2$. For this reason the allies first used the sodium salt of phenol C_6H_5ONa in their early gas masks for the absorption of phosgene. "Urotropine" or hexamethylenetetramine $(CH_2)_6N_4$ was also used. Soda lime is better. It is more effective and much cheaper.

Phosgene dissolves readily in various hydrocarbon oils as benzene C_6H_6, toluene $C_6H_5\,CH_3$, etc.

Rubber is rapidly destroyed by phosgene in the liquid form. Liquid phosgene is also capable of dissolving considerable quantities

of other toxic substances. Thus at 0°C. it dissolves 6.6 per cent of chlorine, and at −15°C., 20.57 per cent of chlorine. Mustard, chlorpicrin, diphenyl chlorarsine, and other toxic compounds will also dissolve in phosgene. Some of these mixtures were used in the past war, especially mixtures of chlorpicrin and phosgene. Chlorpicrin causes irritation and vomiting and it was supposed that this would cause the removal of the mask, when the more toxic phosgene would act. Such mixtures were not very successful but there is always a possibility that more effective mixtures may be devised.

Various devices have been developed for detecting small amounts of phosgene in the air. The British test paper was a strip of paper that had been soaked in the following solution and dried:

5 grams paradimethylaminobenzaldehyde $(CH_3)_2N \cdot C_6H_4 \cdot CHO$
5 grams diphenylamine $(C_6H_5)_2NH$
100 cc. alcohol

On exposure for fifteen seconds to air containing small quantities of phosgene, this paper, which was originally white, turns various shades of yellow to orange, depending on the concentration of the phosgene. It will detect 1 part phosgene per million of air or 0.004 mgm. per liter. In 50 parts of phosgene per million of air 0.207 mgm. per liter, the paper turns a deep orange color in fifteen seconds. Chlorine and hydrochloric acid also act on this paper so that it is not a specific test for phosgene. Nevertheless such tests are very valuable to determine when it has become safe to remove the masks. This paper must be preserved in an air-tight container which protects it from light as this will also affect it.

A lamp may also be used for testing for the presence of phosgene. Alcohol is used for fuel and a copper wire is suspended in the flame. The air is drawn in at the base of the lamp around the flame and the copper wire. If phosgene is present to the extent of 0.3 mgm. per liter of air, the flame acquires a very distinct green color.

Palite. When methyl alcohol reacts with formic acid, the ester methyl formate, results:

$$CH_3OH + HCOOH = HCOOCH_3 + H_2O$$

When this product is treated with chlorine, two atoms are introduced one in the methyl group and one in the formate group, thus producing chlormethyl chloroformate ($ClCOOCH_2Cl$) which was used by the Germans under the name K-Stoff and later by the French under the name Palite. It is a yellow liquid boiling at 105°C.

The effects of palite are very similar to those of phosgene. The gas

is heavier and less easily dissipated. However phosgene is approximately three times more toxic.

Superpalite-diphosgene. When methyl formate is completely chlorinated, trichlormethyl chloroformate results ($ClCOOCCl_3$). This compound is an oily liquid, boiling at 128°C. It was commonly erroneously called diphosgene because its condensed formula $C_2O_2Cl_4$ is double that of phosgene $COCl_2$. However there is no chemical relationship between phosgene or carbonyl chloride and superpalite or trichloromethyl chloroformate. Superpalite is also considerably heavier than phosgene, is of about the same toxicity as phosgene and is in addition an intense lachrymator. Aside from lachrymation, the symptoms produced by superpalite are practically the same as those of phosgene. Both palite and superpalite produce pulmonary edema and to this their toxic effects are due.

CHLORPICRIN

Chlorpicrin or CCl_3NO_2 may be considered as being derived from chloroform by replacing the hydrogen atom by a nitro group, which would make its chemical name trichlornitro methane, or nitrochloroform. The name chlorpicrin was given it because the compound is actually prepared commercially by the action of chlorine on picric acid.

Pure chlorpicrin is a nearly colorless, oily liquid differing little in appearance from water. The commercial product has a slight yellow color due to impurities. The odor is characteristic but not unpleasant. In the field it would probably be detected first by its irritating effect on the eyes.

Chlorpicrin boils at 112°C. and freezes at −69.2°C. It is very slightly soluble in water; 100 cc. of water dissolving only about 0.14 gram of chlorpicrin between 0° and 26°C., while 100 cc. of chlorpicrin dissolves only 1 cc. water at 26°C. Chlorpicrin is a heavier than water and when mixed with water will form a separate layer under it. The specific gravity of chlorpicrin is as follows:

TEMPERATURE	SPECIFIC GRAVITY
°C.	
0	1.6923
10	1.6751
20	1.6576
30	1.6399
40	1.6221

The density of chlorpicrin vapor may be obtained by dividing its molecular weight (164.4) by the volume occupied by one mole of the gas under standard conditions (22.4) which equals 7.35. This divided by the weight of a liter of air under similar conditions (1.286) gives a density of 5.7 for chlorpicrin as compared to air. This is not absolutely exact because chlorpicrin is not a true gas and does not follow the gas laws exactly, but it is a close approximation. Chlorpicrin vapor is evidently heavy enough to displace the air in all depressions in the surface of the terrain.

As chlorpicrin has a boiling point somewhat higher than that of water, its vapor pressure is correspondingly lower. Thus when a chlorpicrin shell bursts, the contents are sprayed into the air and considerable of the liquid evaporates at once. The rest falls to the ground and evaporates somewhat more slowly than water, but at a speed depending upon the temperature.

From the vapor pressure, the maximum concentration of the gas obtainable may be calculated as follows:

TEMPERATURE	VAPOR PRESSURE	CONCENTRATION POSSIBLE IN AIR PER LITER
°C.	*mm.* Hg	*mgm.*
0	6.9	57.5
10	10.8	104
15	14.1	136
20	16.9	184
30	30.5	295
50	80.7	748

Since exposure to a concentration of 2 mgm. per liter for fifteen minutes or less, is lethal, it is evident that there is a wide margin between the possible concentration and the lethal dose.

Mixtures of chlorpicrin and phosgene were frequently used. Thus in a mixture of 75 per cent chlorpicrin and 25 per cent phosgene, the phosgene increases the toxicity of the chlorpicrin. A mixture of 25 per cent chlorpicrin and 75 per cent phosgene will produce lachrymation and nausea in addition to the effects of the phosgene. If sufficient of this gas is inhaled before the mask is applied, the nausea and vomiting produced prevents the subsequent use of the mask, and the more toxic phosgene can produce its lethal effects.

Chlorpicrin is a stable compound. It is not hydrolyzed by water and is unaffected by mineral acids, or by aqueous solution of sodium

hydroxide or ammonia. When mixed with sodium sulphite, two chlorine atoms in the chlorpicrin become replaced by two SO_3H groups and the third chlorine atom is replaced by a hydrogen atom, because sodium acid sulphite is produced by the partial hydrolysis of the sodium sulphite, thus:

$$Na_2SO_3 + H_2O = NaHSO_3 + NaOH$$

This reaction expresses the equilibrium existing in the sodium sulphite solution. The reaction of chlorpicrin and sodium sulphite may be expressed as follows:

$$CCl_4NO_2 + 3NaSO_3 + H_2O = CHNO_2(SO_3Na)_2 + 3NaCl + NaHSO_4$$

This reaction occurs so readily that it is used to determine the concentration of chlorpicrin in the air. A definite quantity of the gas and air mixture is passed into a neutral solution of sodium sulphite which is shaken until the gas is entirely absorbed, and the quantity of sodium chloride formed is determined. From this the amount of chlorpicrin in the quantity of air examined can be calculated readily.

This reaction might also be used as a method for destroying chlorpicrin splashed on equipment, or accidentally spilled during transportation or storage. For this purpose the sodium sulphite solution should be about 50 per cent in alcohol in order to bring about intimate contact between the chlorpicrin and the sodium sulphite. Equipment can be wiped off with a rag soaked in such a solution to remove the gas, but caution should be used in applying this solution to clothing as it is very apt to decolorize fabrics.

RELATIVE TOXICITY

For thirty-minute exposures, lethal concentrations are as follows:

	CHLORINE	PHOSGENE	CHLORPICRIN
Milligram per liter.....	3.0	0.36	0.8

From this it will be seen that chlorpicrin is approximately four times more toxic than chlorine, while phosgene is practically ten times more toxic than chlorine.

SYMPTOMS OF CHLORINE POISONING

Inhalation of low concentrations of chlorine (1:10,000) causes active irritation of the upper respiratory passages bringing on coughing, and a sensation of suffocation. In a strong concentration of chlorine, death is produced very rapidly. The irritation may cause syncope and unconsciousness, by reflex action. Post-mortem examination of men found dead after passage of a wave has shown that some have no pulmonary lesion. This, however, is unusual. In other cases men surprised by cloud gas feel a sensation of intense suffocation, make a deep inspiration, fall, cannot get up, struggle a few moments and die. In the bodies of such cases, the face is blackish, the lips a greenish blue, and the skin has a greenish tint. If necropsy is performed immediately, the peripheral vessels do not bleed. The lungs are retracted, not occupying more than a third of the chest. Their color is greenish gray and their appearance and consistence that of india rubber. On section the lungs are dry, bloodless, and friable, and the same greenish gray appearance as outside. The trachea and bronchi are dry and gray. The heart is dry. The right heart is dilated and contains thick blood which stains the hands brown and is slightly acid. The other organs are normal. In such cases the lungs are so altered that they are incapable of expansion and death occurs promptly by arrest of respiration.

In the majority of cases, exposed to lower, but still toxic concentrations, the course of the disease is different. The first symptoms are the same, burning in the throat, cough and a feeling of suffocation. Dyspnea is intense; speech is impossible, and the man struggles with all the force of his accessary respiratory muscles against asphyxia. Even though he now reaches fresh air the condition will run its course, and the man will die in from twenty minutes to some hours, or even twenty-four hours. In these cases death is due to acute pulmonary edema. As pulmonary edema develops, the shortage of oxygen causes headache, a sense of weakness in the legs, and great lassitude. Even in the milder cases the lassitude and fatigue render these men incapable of fighting.

On examination, the sub-crepitating râles of pulmonary edema may be heard all over the chest wall, together with bubbling bronchial râles. The typical case is cold, with a subnormal temperature, and a slow full pulse. The face is cyanosed and the posture is that of one gasping for breath. The respirations are jerky, often numbering

40 a minute, associated with choking cough and expectoration of a frothy or yellow serous fluid. This fluid was sometimes so abundant that it ran from the patient's mouth when the stretcher was tilted head downwards. As much as 30 to 40 ounces of this fluid was often expectorated in twenty-four hours. This first asphyxial stage may last from several hours up to thirty-six hours, and as the pulmonary edema then subsides, is followed by the inflammatory stage, with the usual symptoms of bronchitis. There is a greenish mucopurulent expectoration, high temperature, and a small rapid pulse up to 160 while the respirations are shallow and fast (60). Diminished expansion is one of the most pronounced physical findings at this time, perhaps due to contraction of the bronchial tubes. Headache and asthenia are marked and may last for several weeks. Sometimes gastro-intestinal symptoms are marked, consisting of epigastric pain, nausea, vomiting, (sometimes blood stained material), and diarrhea. In general, those who survive forty-eight hours, usually recover. The symptoms of bronchitis may persist for several weeks, and there may be some permanent pulmonary emphysema. The heart may remain irregular for some time. It is slow and regular at rest, but accelerates to 100 or 120 as the result of moderate exercise. Other symptoms, as the asthenia may last for several weeks, depending upon the severity of the case. But as experience has shown that ultimately almost all cases make a complete recovery, patients should be cheered by this information. The gloomy prognoses made early in the war when the effects of gas were little understood were undoubtedly provocative of neurasthenia and "pensionitis."

PATHOLOGY OF CHLORINE POISONING

The pathology of all gassed cases varied greatly with the length of time the patient lives after gassing, but changes after pulmonary irritants become more marked as death is delayed up to twenty-four hours. The changes found in cases dying during the twenty-four hours after gassing are as follows:

Body. As a rule the eyes show the redness and discharge of acute conjunctivitis. A frothy fluid exudes from the mouth. Post-mortem changes of the body come on early and may be marked.

Abdomen. The abdominal organs and especially the liver are greatly congested, and the vena cava and its branches are engorged.

Thorax. The lungs are very voluminous, and often overlap in the median line. The pleural surface is stretched and glossy; the lungs

are firm in consistency, and do not collapse, and may show the imprint of the ribs. There is usually an excess of fluid in the pleural cavity and in the tissues of the mediastinum, often amounting to several hundred cubic centimeters.

As the trachea and bronchi are opened, frothy fluid pours out. The mucous membrane of the trachea is red and the vessels deeply injected; later there is a sticky membranous exudate. The lungs retain their shape after removal, and are generally filled with fluid. They are doughy in consistency, and almost entirely non-air-containing. The surface is mottled, having a general purple hue with depressed dark patches of atelectasis, and more numerous raised pink patches of emphysema scattered over the surface. Sub-pleural hemorrhages frequently occur. When the lung is sectioned a large amount of fluid escapes as the edema is tremendous. Perivascular edema is prominent, the adventitia of the vessels being markedly distended with edema fluid. The cut surface is red, translucent and has a gelatinous consistency. The blood vessels are dilated, and filled with blood. During the latter part of the first twenty-four hours small patches of broncho-pneumonia are sometimes encountered.

The heart is generally markedly dilated. All chambers of the heart are dilated, but generally dilatation is more marked on the right side which often projects beyond the left. The endocardium is generally normal but may present red spots caused by subendothelial hemorrhages.

Microscopic changes. The mucous membrane of the trachea and larger bronchi is necrotic, and often separated so that pieces are found loose in the trachea. Later it may be completely sloughed away. In the earliest cases there is no cellular reaction, but after about twelve hours leucocytes and fibrin gather, and we have a typical picture of coagulation necrosis. The acute inflammatory reaction often involves the entire wall of the bronchus, and extends into the peribronchial tissues. The changes in the smaller bronchi are similar in character but less pronounced. There are numerous dilatations and constrictions of the small bronchi, which are not supported by cartilage. These abnormalities in the calibre of the bronchi, together with the plugging by exudate, probably account for the patches of atelectasis and emphysema.

In the lungs are patches where the alveolar walls are mostly ruptured forming large air sacs with the remains of the alveolar septa

plainly visible. These correspond to the light colored emphysematous areas found in the gross specimen. In the remainder of the lung the alveolar spaces are normal or even reduced in size, and filled completely with edema fluid, which coagulates when the tissue is hardened and remains in situ, and sometimes resembles the colloid material of the thyroid, and at other times presents a granular appearance. In some cases detached epithelial cells from the alveolar wall may be found lying in this exudate. The capillaries in the alveolar walls are engorged with blood. Fibrin is abundantly deposited on both sides of the alveolar walls, with strands running across which obstruct the blood flow in the capillaries. It is this obstruction to the circulation which is thought to be the principal factor in causing the dilatation of the right side of the heart, with the subsequent acute passive congestion of the abdominal viscera. While occasional small areas of broncho-pneumonia are encountered during this period, real pneumonic consolidation is a later change. There are no red blood cells in the alveoli except in the areas of hemorrhage. There is, however, a migration of polymorphonuclear leucocytes, which is apparently general, and not necessarily a pre-pneumonic process. Small areas of focal necrosis of the alveolar walls are often seen.

The essential lesions leading to death within twenty-four hours from chlorine poisoning may be summarized as follows:

1. An acute tracheitis and bronchitis progressing to sloughing of mucous membranes.

2. Congestion and edema of the entire respiratory tract. The large areas of pulmonary edema in the lungs alternate with patches of acute emphysema and atelectasis.

3. Acute dilatation of heart followed by a passive congestion of abdominal viscera.

In case of delayed deaths or deaths occurring at various intervals after twenty-four hours, the most important feature is the superadded inflammatory process. Death in this subacute stage is generally due to pulmonary infection, with bronchitis and pneumonia. If the patient lives long enough, this inflammatory exudate tends to be largely resolved, but there is also a strong tendency to organization resulting in a certain amount of fibrosis. The end results of gassing in cases that recover will be discussed elsewhere.

PHYSIOLOGICAL ACTION OF CHLORINE

The progressive action of chlorine may be summarized as follows:

1. Narrowing of bronchioles by spasmodic contraction of muscles in their walls.
2. Obstruction of bronchial tubes by exudate.
3. Flooding the pulmonary air sacs by serious effusion.
4. Disruptive emphysema of lungs, and subcutaneous emphysema of neck.

1. *Spasm of muscular coats of bronchi.* Chlorine is an intense irritant. Its first action when breathed into the lungs is that of a stimulant to the sensory nerves. The sensory nerves are very violently stimulated by the action of the gas producing severe pain. This produces a spasm of the muscular walls of the bronchi, thus narrowing their lumen and producing gasping for breath. The irritation of the larynx, trachea and bronchi produces constant coughing. After a time, the muscular spasm relaxes and breathing becomes easier, but the coughing continues.

2. The violent irritation of the mucous membrane produces an inflammatory reaction, and in many areas the epithelium is devitalized. An inflammatory exudate is poured out, and later pieces of mucous membrane may slough off. All of this causes an obstruction of the bronchi, and acts as a factor in the production of asphyxiation.

3. *Pulmonary edema.* When chlorine first enters the respiratory tract, the spasm of the bronchi and bronchioles prevents the gas from reaching the depths of the lungs. But as we have seen this spasm relaxes after a while, and the chlorine will then reach the alveoli. Here it causes a similar destruction of the alveolar epithelium and an intense irritation, which results in a serous effusion filling the alveoli and making the flooded portions of the lung useless for the gaseous exchange of respiration.

4. The continuous coughing ruptures many of the alveolar cells and air passes out into the tissue of the lungs, and may pass up the hilum of the lung into the neck producing a subcutaneous emphysema of the neck.

The result of all these conditions is interference with respiration and a degree of asphyxia depending upon the severity of the gassing and the length of time elapsing after gassing. Thus a case may pass through these phases and suffer from asphyxia in from two to twelve hours.

SYMPTOMS OF PHOSGENE POISONING

The symptoms of phosgene poisoning are quite different from those produced by chlorine. Even in high concentrations phosgene causes less irritation of the upper respiratory passages, and after passing into the open air, the victim may show few symptoms of poisoning. But after a shorter or longer time, the symptoms of pulmonary edema appear. An example of this delayed action following inhalation of almost pure phosgene, is given in the following account taken from the "Official History of the War," Volume II, London, 1923.

February 3, 1917: A chemist was working at a new chemical product. A syphon of phosgene, required for the synthesis of this substance, burst on his table at 1:00 p.m. A yellowish cloud was seen by a second person in the room to go up close to the chemist's face, who exclaimed, "I am gassed," and both hurried out of the room. Outside, the patient sat down on a chair, looking pale and coughing slightly.

2:30 p.m.: In bed at hospital, to which he had been taken in a car, having been kept at rest since the accident. Hardly coughing at all; pulse normal. No distress or anxiety and talking freely to friends for over an hour. During this time he was so well that the medical officer was not even asked to see the patient upon admission to the hospital.

5:30 p.m.: Coughing, with frothy expectoration, commenced, and the patient was noticed to become bluish about the lips. His condition now rapidly deteriorated. Every fit of coughing brought up large quantities of clear, yellowish frothy fluid, of which about 80 ounces were expectorated in one and a half hours. His face became of a grey, ashen colour, never purple, though the pulse remained fairly strong. He died at 6:50 p.m. without any great struggle for breath. The symptoms of irritation were very slight at the onset; there was then a delay of at least four hours, and the final development of serious edema up to death took little more than an hour, though the patient was continually rested in bed.

In Field Hospitals patients who present no serious symptoms on arrival may leave their beds to visit the latrine, and a moment after returning they may be taken with progressive dyspnea, which in the absence of immediate treatment may be followed by cyanosis and death. Attempted operations, even without anaesthesia, have resulted in sharp aggravation of the patient's condition, and even in sudden death due to acute asphyxia.

The cases developing pulmonary edema to a serious extent fall into two groups.

1. *The blue type.* Asphyxia is marked, and as a result of venous engorgement the face is deeply cyanosed. The lips and tongue are

a full blue color, and there may be visible distention of the superficial veins of the face, neck or chest. There is usually a considerable increase in frequency of respiration in the effort to obtain more oxygen. Cough may be present, and large quantities of serous frothy fluid may be expectorated. The pulse rate is usually a little over 100 per minute and is full and of fair tension.

2. *The gray type.* Cases in this group present an ashen pallor, the lips being pale and leaden colored, and they are in a general state of collapse. Respiration is rapid but shallow. Although much fluid is present in the lungs there is little cough and expectoration. The pulse is very rapid, 130 to 150 per minute, weak and irregular. Blood pressure is very low. These are the cases in which the heart has been dilated to the point of inefficiency, and the prognosis is consequently bad. Most of the fatal cases pass through this stage before death. Four-fifths of the deaths occur in the first twenty-four hours. Very few die after the third day.

Expectoration is a variable factor. In acute pulmonary edema there may be a flood of serous, foaming, blood tinged liquid. This pink froth streams down the mouth and nose until a mushroom-shaped collection of bloody froth is found on the face of the dying man. In some cases, however, which on necropsy show massive edema, expectoration does not occur.

During the course of pulmonary edema the sputum is characteristic. Three distinct layers may be seen:

1. An upper layer of froth.
2. A middle serous layer.
3. A bottom layer of mucilaginous liquid of the consistency of apple jelly, which adheres to the container, and which may be rusty or streaked with blood.

Later as bronchitis develops, the sputum consists of homogeneous masses of muco pus. At this stage or after the development of broncho-pneumonia, numerous bacteria appear, and pneumococci and streptococci may be abundant.

PATHOLOGY OF PHOSGENE POISONING

This cannot be well determined from human cases, because in the field a mixture of gases was commonly used. However the following has been observed. Whenever pulmonary edema was marked, the pleural cavities generally contained an excess of fluid. The lungs on removal from the thorax were voluminous and heavy. In

Lungs of Dog Dying 12 Hours after Phosgene Gassing. Extreme Oedema with Patches of Acute Emphysema

typical cases the average weight of the right lung was 1000 grams, and of the left lung, 875 grams. The lungs may be uniformly bluish red in color or may show petechial hemorrhages and alternating patches of acute emphysema. On section of the lungs, frothy serous fluid drips abundantly from the cut surface. Irregularly alternating patches of edema and acute emphysema are found. The trachea and bronchial tubes are generally filled with thin yellowish or blood tinged frothy fluid. There is little or no inflammatory change in the mucous membranes of the larynx, trachea and bronchi.

The veins are engorged, and the heart, especially the right side is dilated. Petechial hemorrhages are often found beneath the endocardium. Pericardial fluid may be increased in amount.

The abdominal viscera show general venous engorgement and passive congestion. The meninges and brain are also congested, and in cases where death occurred after several days of persistent cyanosis, innumerable small petechial hemorrhages were found throughout the whole of the white matter giving it a speckled appearance. The pathological changes can be determined better on animals. The period of acute deaths from phosgene extends over forty-eight hours.

The pathology of cases dying within this period may be summarized as follows:

Body. Frothy fluid, clear or blood tinged escapes from the mouth and nose. So great has been the loss of fluids that a comparison of the weight of gassed dogs coming to autopsy with the weight before gassing often reveals the fact that there has been a loss of 2 or 3 pounds, even in acute cases. Post-mortem changes are rapid.

Abdomen. Engorgement of the vessels and congestion of the abdominal organs is quite constant. The liver is greatly enlarged and congested. Other organs are congested but not noticeably enlarged.

Thorax. The larynx, trachea and bronchi are filled with fluid, but the mucosa of the trachea and larger bronchi is normal except for some slight reddening. The mucous membranes of the smaller bronchi show more congestion. The lungs are very voluminous and heavy, completely filling the chest and often entirely covering the heart. They are mottled, the bulk of the lungs being of a dark red alternating with pinkish patches. The red areas are glistening, gelatinous in consistency and pit on pressure. They are the areas filled with edema fluid. The pinkish areas may be slightly raised,

are crepitant and may be recognized as areas of acute emphysema. On cutting the lungs fluid escapes in large quantity, and the cut surface is generally uniformly smooth and moist.

The heart, as is the case with the other respiratory irritants, shows no changes due to the direct toxic action of the gas. It does, however, show changes secondary to the pathological process in the lungs, the entire heart being dilated, especially on the right side. Subendocardial hemorrhages are frequent. Otherwise the heart is normal.

Microscopic changes of importance are found only in the lungs. The alveoli nearly everywhere are filled with edema fluid. Considerable desquamated alveolar epithelium, often mixed with a varying number of leucocytes and red cells, may be found in the interior of the alveoli, and in most places, the capillaries surrounding the alveoli are tortuous and distended with blood. Much fibrin can be demonstrated in the alveoli and in the alveolar walls, crossing the capillary beds. Here again we have an abundant anatomical basis for the obstruction to the pulmonary circulation, with the consequent dilatation of the right side of the heart, which, in its turn, leads to passive congestion of the abdominal viscera. Scattered emphysematous areas are found where the alveoli are not filled with fluid. Here the walls of the alveoli may be stretched and sometimes ruptured.

Thus the important acute changes caused by phosgene poisoning are practically limited to the lungs. The upper respiratory tract is unaffected. This is in marked contrast to the action of chlorine. The intense edema and congestion of the lungs, with the concomitant deposition of fibrin, is the primary cause of cardiac dilatation and congestion of other organs.

Later there may be an inflammatory reaction in the bronchioles which extends into the alveoli, and results in patches of bronchopneumonia. But the majority of cases recover if they survive the first forty-eight hours.

In order to study the development of pathological changes from the beginning of gassing, it is necessary to gas a large series of animals, killing them at various intervals after gassing. This work has been carried out by several observers. Dunn used goats, Edkins and Tweedy used rabbits and Laqueur and Magnus used cats.

There is usually no perceptible change in the naked eye appearance of the lungs less than half an hour after gassing by moderate

concentrations. By the end of half an hour, it is often found that the centers of the lobules on certain areas are marked out as red areolae. This is due to the commencement of alveolar edema, and the redness arises from the fluid rendering the tissue more transparent, so that the color of the blood vessels is brought more clearly to the eye. These first traces of alveolar edema are soon accompanied by the development of edema in the interstitial tissues. On histological examination, alveolar edema is always recognizable at the end of half an hour, as masses of light granular material, found practically throughout the lungs in the alveolar cavities. Damage to the capillary blood vessels in the infundibular and alveolar walls may also be recognized at the end of half an hour after gassing. In the earliest recognizable stages the damaged vessels appear collapsed and contain few red blood cells, but many leucocytes. As time goes on, minute thrombi form in the capillaries lining the peripheral alveoli of the infundibulum and these thrombi thicken and may completely block the lumena of these capillaries. On the other hand the majority of the capillaries of the more distal alveoli became enormously engorged and the blood is in a condition of stasis. The visible lesion is therefore an extensive and severe impairment of the capillary network whereby the vessel walls are rendered unduly pervious. The loss of alveolar epithelium, indicated by desquamation, may be a contributory factor in the causation of edema, but it hardly seems possible that loss of epithelium alone would occasion such excessive transudation of fluid as ultimately occurs. During a period lasting several hours the amount of fluid remaining in the alveoli is very small. The early appearance of edema in the interlobular septa is an index of the rapidity with which such abnormal exudate can be disposed of from the alveoli. Drainage of fluid by the lymphatics and bronchi occurs more readily from the more elevated situations. But ultimately, after several hours, large areas become filled with fluid to the exclusion of all air. The amount remaining in the alveoli represents the degree to which supply is in excess of disposal, and varies greatly in different animals. Thus while some animals reach a fatal condition in twelve hours with lung-heart ratios of 1:3 to 1:4, others killed at twenty-four hours have much higher ratios, and edema not yet fully developed.

THE PHYSIOLOGICAL ACTION OF PHOSGENE

In animals, inhalation of concentrated phosgene causes a change in respiratory movements and a marked contraction of the bronchial muscles. Immediately after the first breath the lungs become a mottled brownish color on the upper surface. In the course of a few minutes the brown specks increase in number and size. The respiratory movements become progressively smaller and finally almost imperceptible. The circulation falls precipitously. When tested with litmus paper the brown spots of the lung are distinctly acid. If these lungs are examined microscopically it is found that the large blood vessels are markedly contracted and in other places the lumen has entirely disappeared. The bronchi are markedly contracted, the lumen is stellate and the mucosa gone. The alveoli are empty, the tissues are swollen, and the capillaries are filled with agglutinated, colorless old blood corpuscles. There is no edema fluid. That these injuries must result in quick death is obvious.

Death from phosgene in concentrated form is evidently quite different from the death resulting from inhalations of moderate or low concentrations. In the former the damage is so intense that the lung abruptly ceases to function, and the shock causes circulatory failure also. Death occurs in a matter of several minutes. But with moderate or low concentrations there are no immediate symptoms and death finally occurs as the result of the gradual development of pulmonary edema.

In such concentrations, the action of phosgene differs from that of chlorine. Phosgene causes practically no irritation of the trachea and bronchi and subjective irritation is much less in evidence. Coughing is less, and disruptive emphysema is practically never seen. Moreover the results of gassing are more insidious in their action. After exposure to moderate concentrations of phosgene a man may feel able to carry on his work for an hour or two with only trivial symptoms, but he then becomes suddenly rapidly worse, presents a picture of extreme cyanosis which later passes into a greyish-white stage of collapse which is rapidly fatal. Thus men who have passed through a gas attack and seem to have suffered but slightly, have died suddenly some hours later upon attempting some bodily effort.

Precisely similar effects are observed when animals are gassed with moderate concentrations. Under these circumstances there are few symptoms during or immediately after gassing. There is little coughing. The respirations may become more frequent and shorter

than normal. There is no protective reflex, i.e., contraction of the mucosa of the upper air passages. From the start the gas is inhaled into the depths of the lungs, and here its harmful effects develop. After the conclusion of gassing, animals usually show no symptoms for several hours. After two to six hours dyspnea begins. The labored breathing becomes gradually more marked and at the height of the illness, the respirations may reach 80 to 100 per minute. Death during the acute stage (twenty-four to forty-eight hours) is due to pulmonary edema. The pathological condition produced in the lungs as the result of the irritant action of phosgene, has already been described. The condition that we have to treat may be summarized as follows: After inhalation of phosgene at moderate concentrations, the development of pulmonary edema begins very early, in some animals within two hours. The edema is at first non-cellular, but after about five hours a few leucocytes are found and later the exudate may be rich in cells. In phosgene poisoning, red blood cells are seldom found in the exudate while after inhalation of chlorine, erythrocytes appear in great numbers. Later fibrin appears and there are foci of broncho-pneumonia. But at the height of the illness the picture of edema is most prominent. After the second or third day if death has not occurred, the fluid is resorbed and recovery will ensue barring complications due to broncho-pneumonia.

Since the irritant action of phosgene, in concentrations that will be met in the field, upon the external mucous membrane is so slight, and the entire clinical picture is due to the changes produced in the lung tissue and the secondary effect of these changes, namely, progressive pulmonary edema, we may well endeavor to understand the physiological action of phosgene. If we can explain the action of phosgene and learn to treat it, we will have a basis for the treatment of the pulmonary edema produced by any other pulmonary irritant.

THE PATHOLOGICAL PHYSIOLOGY OF PHOSGENE POISONING

1. The production of edema

The important acute changes caused by phosgene poisoning are practically limited to the lungs. Studying these changes from the beginning, it will be found that damage to the capillaries may be demonstrated in sections as early as half an hour after gassing. At

first the capillaries in the walls of the alveoli are markedly constricted and appear collapsed. Later they become dilated, much engorged, and there is evidently a condition of stasis. Often minute thrombi form and block the capillaries for some distance, which undoubtedly increases the tendency to stasis. This dilatation of the capillaries with stasis is the main cause of the pulmonary edema, which progresses rapidly from this time on. Alveolar edema may be demonstrated in animals killed half an hour after gassing and thereafter it increases in amount.

In a condition of capillary stasis, Krogh and others have shown that there are actual breaks in the continuity of the cells composing the capillary walls. Through these minute gaps between the cells, the blood plasma ooozes out. It collects in the alveoli because the fluid is poured out more rapidly than it can be carried away by the periarterial lymphatics. The fluid will continue to increase until exudation is less than absorption by the lymphatics. When the collection of the edema fluid reaches a sufficient amount, the gaseous exchange becomes so diminished as to produce serious anoxemia, and the symptoms of anoxemia set in. Until this time there are few symptoms of importance. We have here therefore the reason for the latent period in phosgene poisoning which is ordinarily several hours. After from two to six hours dyspnea begins, and the labored breathing becomes gradually more marked until at the height of the illness, the respirations may reach 80 to 100 per minute.

The mechanism of the production of pulmonary edema. There are a number of theories concerning the production of edema which have been classified essentially as follows:

1. Obstruction to lymph outflow.
2. Increased blood pressure.
3. Decreased extravascular pressure.
4. Increased permeability of the capillary walls.
5. Increased filterability of the blood plasma.
6. Osmotic pressure changes—extravascular increase or intravascular decrease.
7. Changes in affinity of colloids for water.

Pulmonary edema has been produced experimentally by a variety of measures fulfilling one or more of the above theoretical requirements. These measures include the production of intravenous emboli; intravenous injection of drugs like adrenalin and muscarin, and toxic chemicals such as silver nitrate and collargol; injury to the

chambers of the left heart; ligations and compressions of aortic and pulmonary branches; occlusions of bronchi and bronchioles; alterations in the blood including infusions of large amounts of fluid; increasing the venous inflow beyond the maximum cardiac output; decreasing oxygen supply to the lung parenchyma.

We may thus see that pulmonary edema may be produced in a variety of ways, and we must decide which ways are operative in the case of phosgene.

Method of the production of edema by phosgene. Conclusive proof may be afforded that the edema depends upon the local injury to the alveolar capillaries and that other changes in blood and circulation are secondary to this.

In phosgene poisoned lungs, edema sets in at a perfusion pressure of 30 to 40 mm. Hg. But in normal lungs no edema is produced even if the pressure is increased to 82 mm. Hg. Again venous obstruction of such intensity that the blood stream becomes stationary produces no edema as long as the arterial pressure does not exceed 35 mm. Hg. In phosgene lungs on the contrary a pressure value that is normal or only slightly above, and a very moderate reduction of venous flow, produce edema. Indeed even a simple arterial increase may produce edema. By the addition of ammonia to the blood stream, the lung tissue may be so damaged that even with normal circulation, edema occurs. It is evident that in phosgene poisoning the permeability of the pulmonary capillaries for fluids (plasma) is increased.

Another cause for the edema is undoubtedly to be sought in the acid produced in the tissues by the decomposition of phosgene. The tissues damaged by phosgene are intensely acid. Now Fischer's theory of edema is that

> A state of edema is induced whenever, in the presence of an adequate supply of water, the affinity of the colloids of the tissues for water is increased above that which we are pleased to call normal. The accumulation of acids within the tissues brought about either through the abnormal production, or through the inadequate removal is chiefly responsible for this increase in the affinity of the colloids for water. Any chemical means by which we render possible the abnormal production or accumulation of acids in the tissues is accompanied by edema.

Pulmonary edema also results whenever the oxygen supply to the lung is sufficiently interfered with because the lack of oxygen leads to the accumulation of acids in the tissues. It is easy to produce edema in the excised lung because the normal oxygen supply is cut off.

Fischer took sheep lungs and permitted water or physiologic saline solution to enter the pulmonary arteries, and thus produced an intense edema. A 500-gram lung under these circumstances will take up several liters in an hour. Two periods can be designated. During the first period there is no fluid in the trachea. Later the pleural surface is moist and a bloody frothy fluid emerges from the trachea. Although the veins are not ligated no fluid escapes from them. The longer the lungs have been out of the animal, the more quickly does pulmonary edema appear.

In phosgene poisoning we have, therefore, a vicious cycle. The increased capillary permeability and the intense acid reaction produced by the hydrolysis of phosgene in the tissues, results in prompt development of pulmonary edema. This edema results by its mere presence, as well as by the thickening of the blood, the cardiac embarrassment and other mechanical factors, in an almost insuperable difficulty in obtaining oxygen. This results in further acidic changes in the lungs producing further edema.

2. Changes in the blood

1. *The primary dilution.* That the edema fluid is simply blood plasma may be shown, not only by the pathological mechanism explained above, but by chemical examination of the fluid. Its chemical composition is precisely that of blood plasma.

Now since normally the red corpuscles form approximately 50 per cent of the volume of the blood, the degree of dilution or concentration of the blood may be measured by the percentage of hemoglobin. It has been found that immediately following gassing there is a slight but decided fall in the percentage of hemoglobin indicating a primary dilution of the blood. This dilution probably coincides with the period of capillary contraction and may be caused by "plasma skimming." That is, the capillaries are contracted to such a degree that the corpuscles no longer pass through, although the plasma does pass. Thus the corpuscles are dammed back in the lung, and there is an increased percentage of plasma in the peripheral circulation. But this phase soon passes off; the capillaries dilate and plasma is poured into the lung.

2. *The thickening of the blood.* This large quantity of fluid abstracted from the blood causes a great concentration of the blood. This is shown by the increase in hemoglobin and red blood cells. Increases of hemoglobin by the Sahli of 25 to 50 per cent may be demon-

strated in gassed cases both animal and human. The normal hemoglobin by the Sahli apparatus is 80 per cent, while in gassed cases in field hospitals during the first four days it may reach 122 per cent. In the majority of cases a return to normal occurs after the fifth day. At the height of gas poisoning, the erythrocyte count in man was found to be over 9,000,000. The same polycythemia is found in animals. As might be expected if the blood is thickened, the peripheral vessels are markedly contracted and the blood flows feebly at the height of poisoning, so that blood can be obtained from the ear only with great difficulty. In experimental animals it must generally be taken from a vein.

An animal usually has 70 grams of blood per kilo. At the height of the disease, in an average of 23 experiments, 21.3 grams of water were lost from this original 70. In individual cases as much as 34.3 grams per kilo were lost. Such an intense loss of water from the blood in such a short time rarely occurs in other diseases. It is obvious that therapeutic measures must be adopted to compensate for this loss.

In 23 experiments the increase in weight in the lungs owing to edema fluid averaged 23.3 grams per kilo. The loss of fluid from the blood estimated by hemoglobin determinations averaged 21.3 grams per kilo. It will thus be seen that the loss of fluid from the blood corresponds almost exactly with the increased weight of the lungs. This is a complete demonstration of the theory that the edema fluid comes directly from the circulation. The amount of fluid lost from the circulation as compared to the edema fluid in the lungs is the same in man. At autopsy on gas victims it has been found that the weight of the lungs were 2 to 2.5 kilos or more instead of the 500 to 600 grams in normal cases. Since an adult man has from 4 to 5 liters of blood plasma, it is evident that from one-third to one-half of this circulatory fluid has passed out into the lungs.

It might be expected that the fluid thus abstracted from the blood might be compensated by abstraction of fluid from the tissues. That this is not the case in phosgene poisoning to any degree depends on the fact that the fluid in the lungs possesses exactly the composition of the blood plasma, so that there is no possibility for osmotic changes which are the principle cause for the interchange of fluid between the blood and the tissues.

However, experiments have demonstrated that venesection, especially when practiced early, markedly lessens the tendency to pulmo-

nary edema. This beneficent action may be explained as follows: After venesection although the osmotic tension of the blood is not changed, the pressure ratio between the circulation and the tissues is suddenly changed. This leads to a subsequent dilution of the blood from the water in the tissues. In untreated gas poisoning the passage of fluid into the lung takes place gradually in the course of hours, so that no sudden variation of pressure between the blood and the tissues occurs. Venesection makes an abrupt and pronounced change of pressure in the opposite direction. It thus not only halts the increasing pulmonary edema and thereby aids the patient in obtaining oxygen from the air, but by the dilution of the blood which follows, it makes a freer circulation possible and thus reduces the anoxemia of the tissues, and relieves the cardiac embarrassment.

3. Changes in the circulation of the blood

The first changes produced are reflex and practically instantaneous. Mayer in a report December 3, 1915 (Sur les Reactions Reflexes Respiratoires et Circulatoires determines par le passage des gaz et vapeurs dans les premieres voies respiratoires) reached the following conclusions:

1. The arrival of an irritant gas in the upper respiratory tract immediately produces the reflex studied by Knoll and Franck. This reflex expresses itself as follows:

a. By a modification of the respiratory rhythm resulting in spasmodic movements of the organs causing pulmonary ventilation.

b. By a modification of the rhythm and amplitude of the cardiac pulsations.

c. By a modification of the blood pressure.

1. *The pulse.* The marked slowing of the pulse and prompt fall in blood pressure caused by this reflex, and immediately following gassing is observed in practically all experimental animals and is shown on figure 1. Following this reflex slowing of the pulse, there is a reaction which carries the pulse above the normal rate. After more or less violent fluctuations the rate returns to normal. The later gradual rise in the pulse rate occurs during the period when pulmonary edema and its resulting anoxemia is setting it. Thus, Schneider and Truesdell have shown that anoxemia alone, induced by breathing pure nitrogen, accelerates the heart with amazing promptness and that the increase in pulse rate continues gradually as long

as the experiment can be continued. This explains the very rapid pulse in the later stages of gas poisoning and shows how the heart may be relieved simply by administration of oxygen.

2. *The blood pressure.* The course of the blood pressure is very similar. Immediately following gassing there is a pronounced fall in both the systolic and diastolic pressures which is clearly caused by the reflex described by Mayer. Both systolic and diastolic pressures

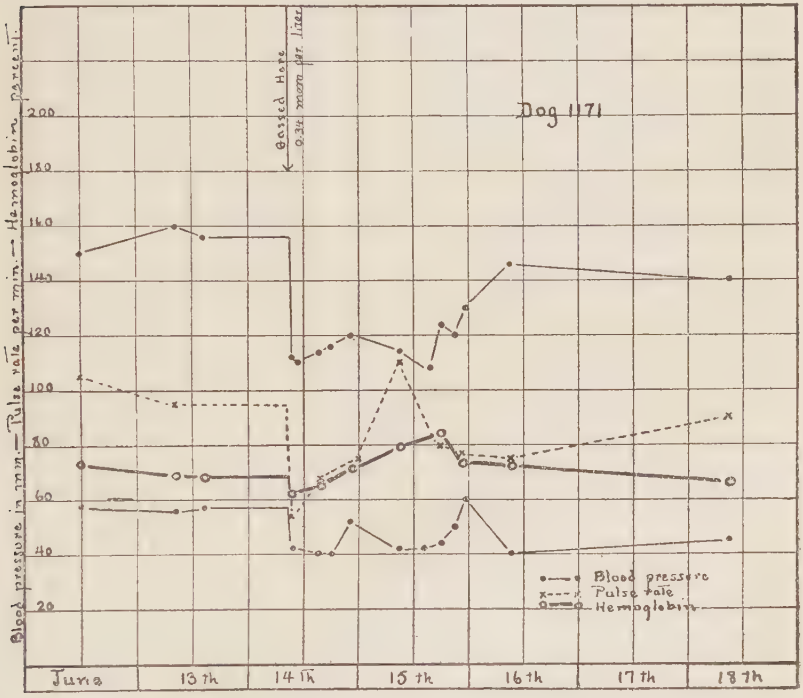


FIG. 1

then return to normal after more or less violent fluctuations. Later as pulmonary edema develops, there is in general an increasing fall of the systolic pressure, accompanied by a more gradual rise of the diastolic pressure. Thus when pulmonary edema is at its height, the pulse pressure is often quite small. This decrease in the amount of blood circulating can only increase the anoxemia already present, for what little oxygen is absorbed from the lungs cannot be carried efficiently to the tissues. This diminishing pulse pressure is to be

explained by the interaction of three factors, a progressive weakness of the heart, injury to the vaso motor centers that follows anoxemia of the brain, and the decreased volume of the blood owing to the loss of its plasma into the lungs.

3. *The pulmonary circulation.* As has already been stated, the arrival of the irritant gas in the depths of the lungs is followed by an immediate and marked constriction of the pulmonary capillaries.

The rapidity of the pulmonary circulation at this time has been measured by the Stewart method which consists in observing the passage in the exposed carotid of colored particles injected into the jugular vein. At the moment of inhalation of gas, a considerable increase in the time of passage through the cardio-pulmonary circulation is observed. This time may be increased from three to twenty seconds.

Subsequent to this primary obstruction, and during the first hour or two after gassing, the flow of blood through the lungs is only slightly lower than normal, and this in spite of moderate edema at this time. With the appearance of extensive edema there is a noticeable blocking of the blood stream through the lungs. In experiments on normal lungs (cats) it has been found that a normal circulation may be maintained by a pressure of 30 mm. Hg. In lungs that had developed edema following gassing with phosgene, practically no circulation could be produced by this pressure. A pressure of 40 mm. Hg or more was necessary. From a number of experiments it was proved that in the normal lung, a pressure of 40 mm. Hg gives about twice as much circulation as 30 mm. Hg.

This diminished pulmonary circulation cannot be explained entirely by the increased viscosity of the blood, because the same blockage exists when the lung is perfused with normal blood. Further in the initial stage of pulmonary edema, when the amount of fluid in the lung is small, the circulation is still good. The blockage must therefore be due chiefly to the high grade of pulmonary edema. Nevertheless at the height of the illness the viscosity of the blood doubtless further reduces the amount of this circulation.

4. *The heart.* The constriction and plugging of the pulmonary capillaries greatly increases the resistance in the pulmonary circulation and the work of the right heart. At this stage the right heart frequently dilates and this dilatation may be demonstrated by X-ray examination. This primary dilatation may be brief or may last several hours depending upon the intensity of the gassing. The

duration of primary dilatation coincides very closely with the duration of the primary dilution of the blood, both being caused by the constriction of the capillaries.

Later when the capillaries dilate, the size of the heart returns to normal. During the development of pulmonary edema, the heart decreases in size. As a similar reduction may be observed after severe hemorrhage, it is probable that the reduced size of the heart is caused by the diminution in the volume of the blood. During the period of asphyxial death, the heart enlarges again, particularly the right side. After death the heart is dilated.

The heart is not directly injured by the phosgene, which is decomposed entirely in the lungs. At post-mortem there are never any noticeable parenchymatous changes in the heart muscle. The effects of phosgene poisoning on the heart are partly mechanical, and partly caused by anoxemia. Thus at the height of phosgene illness there is a series of conditions that injures the heart:

1. Anoxemia. At the height of the illness there is a great deficiency of oxygen. This directly affects the nutrition of the heart as well as the vital centers in the brain.

2. The great increase in viscosity. At the height of the illness the blood is almost tarry in consistency and much more force is required by the heart to force it through the capillaries.

3. In cases of intense dyspnea the amount of muscular work is greatly increased. This produces the same effect as any other exercise. The heart must accelerate.

4. In a state of edema, a blocking of the circulation in the lungs, throwing a great load on the right heart.

All of these conditions work together against the heart, and accordingly the functional ability of the heart is markedly influenced. The right heart usually becomes dilated and weakened. From this description the behavior of the heart in phosgene poisoning is easily understood. In the mild cases, and in the beginning of the severe cases, the pulse is normal, strong and regular. A short time after gassing there may be bradycardia. In the severe cases, the pulse rate gradually increases up to 160 to 180 per minute, and becomes small, irregular and difficult to detect. At the same time percussion and x-ray show a dilatation usually on the right side, but occasionally of the entire heart. This dilatation produces pulsations of the vessels of the neck, and systolic murmurs may be heard. Cases may die suddenly in collapse as the result of failure of the heart.

4. Anoxemia

Anoxemia as a factor in gas poisoning is of three types.

A. The anoxic type in which there is a low pressure of oxygen in otherwise normal blood. This may be caused by deficiency of oxygen in the atmosphere as at great heights, or by interference with the gaseous exchange between the blood and air in the lungs.

B. The hemic type. This is due to the fact that there is a deficiency of hemoglobin, whether caused by hemorrhage, anemia, or combination with some other factor like CO, which prevents hemoglobin from uniting with oxygen.

C. The stagnant type. The blood may have a sufficient supply of oxygen, but it is supplied to the tissues in insufficient quantities.

When an atmosphere devoid of oxygen is breathed, there is an almost instantaneous loss of consciousness. In atmospheres in which the concentration of oxygen is seriously reduced there is a chain of symptoms including mental failure, headache, loss of muscular power and coördination, weakened heart action, and dyspnea. These are the symptoms of anoxemia from whatever cause produced.

The effect of anoxemia on the heart has already been noted, It has been extensively observed in carbon monoxide poisoning. When the hemoglobin is quarter saturated with carbon monoxide, there is no discomfort during rest, but on exertion, dizziness and faintness follow. As the amount of oxygen decreases it becomes less and less possible to make any exertion. The heart cannot respond to the increased work and the blood supply to the brain fails. This diminished capacity is accompanied by a marked increase in the frequency of the pulse without any corresponding increase in the volume of blood expelled. Finally the pulse becomes irregular due to incoördination within the heart.

Cases of phosgene poisoning suffer from anoxic anoxemia because the edema fluid prevents the interchange of gases in the lungs, and also from stagnant anoxemia which has been sufficiently described under the circulatory changes.

1. *The cause of sudden death of gassed cases.* Sudden death following exercise or struggling has often been observed in soldiers in the field and in experimental animals. The cause of these sudden deaths is readily demonstrated. In a normal animal when the corpuscles are saturated with oxygen, the oxygen in the arterial blood fluctuates between 94 and 98 per cent. The oxygen percentage in the venous blood is generally about half that of the arterial blood. Barcroft

found, after gassing a goat at a moderate concentration, that the oxygen percentage in the arterial blood dropped to about 80 per cent. This may cause headache, impaired vision and inability to work, but is by no means dangerous to life. However when the same goat struggled, the arterial oxygen dropped abruptly to 44 per cent. This is caused by the greatly increased consumption of oxygen by the tissues following exercise. Such a high degree of anoxemia affects at once both the cardiac and respiratory centers of the brain. A high degree of anoxemia also stimulates the vagus with the result that when the animal struggles, the heart ceases to beat. This adds stagnant anoxemia to the already high anoxic anoxemia. The respiratory center fails, the animal stops breathing and dies, even should the heart resume beating.

2. *The blue and grey types of anoxemia.* All observers of gassed cases have described two clinical types, the blue or plum colored cases, in which there is deep blue cyanosis, and the grey cases. All observers are agreed that the grey cases are much more serious, and that bleeding does little good if it is not positively counter-indicated. The grey type was seen much more frequently after gassing by phosgene than after chlorine, and Haldane states that practically all phosgene cases develop the grey type of anoxemia. What is the physiologic basis for these two types? This basis is (*a*) that the expansion and contraction of the systemic capillaries depends upon the reaction of the blood, i.e., the relative percentage of oxygen and carbon dioxide; and (*b*) that the affinity of hemoglobin for oxygen depends at least partly upon the percentage of carbon dioxide in the blood. Let us apply these two principles to the blue and grey cases.

The blue or plum-colored cases. In these cases the per cent of oxygen in the arterial blood is seriously diminished, giving the blood its blue color. It is practically venous blood. But the amount of carbon dioxide is increased. This acid reaction causes an active dilatation of the capillaries, and their distention is shown by the color of the lips and skin. The blood therefore passes through the systemic circulation rapidly. The heart is failing because owing to the anoxemia it is improperly nourished and cannot respond to the increased work thrown upon it by this increased flow. The right side becomes distended, and the blood backs up in the veins. Fullness of the superficial veins with the blue type of cyanosis therefore indicates a failing heart. Bleeding is the obvious remedy for this cardiac embarrassment. Of the value of venesection in cases showing venous engorge-

ment and full blue cyanosis there can be no doubt. The abstraction of blood is rapidly followed by diminution of cyanosis, relief of dyspnea, headache and general discomfort. This immediate relief suggests that in the main the treatment owes its effect to the immediate relief of an embarrassed circulation at a critical period when the right side is dilated and beginning to fail. It undoubtedly also causes a permanent dilution of the blood, and hinders exudation of fluid into the lungs or promotes its absorption.

The grey cases. In the grey cases there is also serious anoxemia, but its mechanism is different because the percentage of carbon dioxide is greatly reduced. The superficial capillaries contract and little blood passes through them. These cases are in a much more critical condition than the blue cases because the less the carbon dioxide in the blood the greater is the affinity of hemoglobin for oxygen. Thus if there is less carbon dioxide than usual in the alveolar air, the hemoglobin takes up oxygen more readily and the arterial blood will be less apt to be blue. But also, and this explains the critical condition of the grey cases, there is less carbon dioxide in the blood and the hemoglobin parts with its oxygen to the tissues less readily. Thus in grey cases, although the heart may be weak, and the pulse feeble and rapid, there is no venous engorgement, and the main danger is not from cardiac collapse but from failure of the respiratory center. Bleeding would therefore be useless (except for the purpose of diluting the blood) and perhaps harmful. The administration of oxygen in imperative.

We may now ask what causes this diminution in the amount of carbon dioxide in the grey cases? The answer is contained in the effects of anoxemia on the respiration.

Anoxemia and respiration. The longer the anoxemia lasts, the more serious is the damage to the central nervous system, and recovery may never take place if the anoxemia is sufficiently prolonged. This may be seen in carbon monoxide poisoning which is pure anoxemia. Thus if a man has been unconscious for some hours, he may never recover even though all the carbon monoxide may be removed from his blood in the course of an hour or two. Of all the effects of anoxemia on the central nervous system, the effect on the respiratory center appears first and is the worst. When this center is impaired in function it results in diminishing depth and increased frequency of breathing. Now in the process of expansion of the lungs, the different parts of the lung open in succession not simultaneously. There-

fore in shallow breathing only the upper parts of the lungs will open properly. These will be over ventilated and the lower parts will be under ventilated. The hemoglobin is saturated with oxygen by normal ventilation and therefore no increase of oxygen absorbed can occur by over ventilation of any part. On the other hand the under ventilation of other parts will cause the oxygen percentage in the blood of these parts to fall below normal. The net result will be that the mixture of arterial blood from over ventilated and under ventilated parts will contain less oxygen than usual.

In the case of carbon dioxide, however, the over ventilation of the upper part can remove just as much more carbon dioxide than usual as the under ventilation removes less. Hence at most the mixed arterial blood will contain a normal per cent of carbon dioxide. This will give rise to the blue type case. But the interposition of a thick layer of edema fluid introduces another factor. Carbon dioxide is far more soluble in water than is oxygen and carbon dioxide will pass out through this fluid far more easily than oxygen can pass in. Further any increase in breathing will cause a material lowering of the partial pressure of carbon dioxide in the alveolar air and will facilitate the escape of this gas from the blood. Thus the carbon dioxide of the blood is surely reduced by rapid shallow breathing especially when edema fluid is present in the lung. It is this combination of diminished oxygen and carbon dioxide, that produces the grey cases. The deadly significance of this condition in gas poisoning lies in the fact that rapid shallow breathing increases the anoxemia and in turn the anoxemia affects the respiratory center to produce more rapid and shallow breathing so that if the chain is not broken it can end only in death. The administration of oxygen breaks this sequence. The rate of diffusion of a given gas through a layer of liquid is determined by the solubility of the gas in the liquid and by the difference in the partial pressure of the gas on the two sides of the liquid. We can easily raise the partial pressure of oxygen in the alveolar air by giving the patient pure oxygen to breathe. The oxygen percentage in the blood is thus increased and as the anoxemia diminishes, the respiratory center revives and the breathing becomes slower and deeper, enabling the patient to get still more oxygen. Thus the grey case should be treated first with pure oxygen in order to raise the partial pressure in the alveolar air as much as possible. When the anoxemia has disappeared as shown by restoration of a normal pink color of the mucous membranes, and the respiration has become

slower and deeper, the patient may be able to get sufficient oxygen from breathing a 60 to 40 per cent mixture of oxygen.

SYMPTOMS PRODUCED BY CHLORPICRIN

Chlorpicrin is a rather intense lachrymator, though far less effective in this respect than the true lachrymators. Lachrymation would be the first warning of its use. Chlorpicrin causes more coughing than phosgene, for chlorpicrin produces a true bronchitis, the medium sized and small bronchi being affected most severely. Prominent among the symptoms of chlorpicrin is nausea and vomiting. This may follow gassing by any of the pulmonary irritants, but is far more violent and pronounced in the case of chlorpicrin.

In high concentration it may cause unconsciousness and sudden death, but this is very rare.

In severe cases of chlorpicrin poisoning, asthenia or general muscular weakness is most marked, with a weak, rapid and irregular heart. The patient appears to age rapidly, and there may be an acute nephritis. In this connection it must be remembered that it has been claimed that chlorpicrin is absorbed in the blood stream and acts as a systemic poison, while phosgene and chlorine are not so absorbed. However the presence of chlorpicrin in the blood of gassed animals has not been demonstrated and there are several reasons for believing that it is not so absorbed. Renal congestion and even a certain degree of nephritis has been observed in serious cases of poisoning with both phosgene and chlorine as well as with chlorpicrin. Chlorpicrin has been injected into animals subcutaneously in lethal doses, but the kidneys of animals so killed have been normal.

It seems more probable that the congestion and nephritis following chlorpicrin poisoning are more probably due to the general congestion of the viscera following the damming of the circulation of the lungs and dilatation of the right heart, and to the anoxemia suffered by these tissues as one of the results of the severe general anoxemia. Chlorpicrin causes a very high grade of pulmonary edema, and this is its most serious action.

It has been noted that those who have been injured by chlorpicrin appear to become more susceptible to its action, so that concentrations of the gas which are not noticed by others may cause distressing symptoms.

Chlorpicrin is also irritating externally. Liquid chlorpicrin may even cause ulceration of the skin, and scratches and abrasions exposed to chlorpicrin fumes are prone to become infected with development of abscesses and lymphangitis. Chlorpicrin fumes may cause conjunctivitis and liquid chlorpicrin splashed upon the cornea may cause ulceration and loss of vision.

THE PATHOLOGY OF CHLORPICRIN

Poisoning is similar in some respects to chlorine poisoning and in others more resembles phosgene. The essential lesions consist of:

1. Necrosis of the bronchial epithelium and bronchiolar walls.
2. Extreme edema and congestion of the lungs.
3. Dilatation of the heart followed by passive congestion of abdominal viscera.

Chlorine attacks the epithelial lining of the upper portion of the respiratory tract, the larynx, trachea and larger bronchi. Although the injury proceeds to the alveoli producing pulmonary edema, the edema is never so severe as that caused by phosgene. The most severe injury is suffered by the trachea and bronchi.

Phosgene, which must be hydrolyzed before it can attack the cells, passes over the trachea and larger bronchi and destroys the epithelium of the smaller bronchi and bronchioles, and especially the alveoli, the walls of which are everywhere damaged.

Chlorpicrin occupies an intermediate position in its action on the respiratory tract. The mucous membrane of the trachea and larger bronchi are injured in places but nowhere is the damage as severe and necrotizing as follows the use of chlorine. But the medium sized and smaller bronchi are injured to even a greater extent than with phosgene or chlorine, while the changes in the alveoli and the degree of pulmonary edema is practically the same as after the use of phosgene.

The severe bronchial lesion, including the formation of obstructive casts, is separated by a space from the edematous lesion in the alveoli, and evidently arises independently of it. Because of this blockage of the bronchial tubes, there is a greater tendency to the development of emphysema than is the case in phosgene poisoning, again being more like chlorine in this respect.

Each of these three gases produces an intense edema of the lungs, developing rapidly after exposure. The development is more rapid with chlorine and chlorpicrin than with phosgene. With the same

concentration, the peak of the edema is reached in less than twelve hours with chlorine and chlorpicrin, while after phosgene gassing the peak of edema is reached in about eighteen hours.

DIAGNOSIS

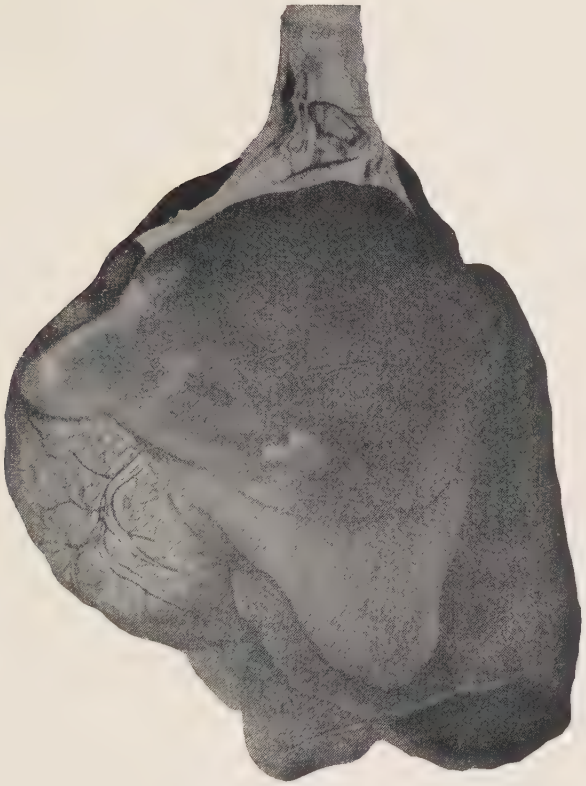
From what has been said with regard to the delayed action of phosgene, it is evident that it will not always be easy to make a diagnosis of gassing. Many men may attempt to leave the front lines alleging that they have been gassed, some because they may really believe it and some because they are malingering. Should the surgeon know that no gas had been used, he can safely return such men to the front. But when gas has been used, it is the part of of wisdom to give any case claiming to be gassed the benefit of the doubt. Dubious cases may be retained at the collecting station. As pulmonary edema reaches its height in about twenty-four hours, at the end of this time it will be possible to discharge any cases that were mistaken about being gassed or who are malingering. An interesting point in this connection is the observed fact that after gassing, the taste for tobacco is lost. The Surgeon may conclude quite safely that a man smoking cigarettes has not been gassed.

At the first examination the man should be asked to describe his own symptoms and no leading questions should be asked. He may have vomited, or complain of weakness and exhaustion. Pallor, change in pulse rate or respirations may be noted. Presence or absence of cough should be noted and whether breathing is painful. Any or all of these symptoms point to the probability of gas poisoning.

TREATMENT. FIRST AID

Loosen clothing and equipment. The first aid treatment consists in keeping the patient at absolute rest in the recumbent position and keeping him warm. The lack of oxygen for the vital tissues, the heart and brain is one of the main causes of a fatal issue.

During quiet breathing, the inflow and outflow of air is about 500 cc. As a man breathes from 15 to 20 times per minute it may be computed that in twenty-four hours a man breathes from 10,600 to 14,400 liters of air. Expired air is 4.78 volumes poorer in oxygen than inspired air and 4.34 volumes richer in carbon dioxide. It will thus be seen that from 516 to 688 liters of oxygen are consumed in twenty-four hours. Moreover this is a minimum requirement. If the



HEART AND LUNGS OF DOG DYING 12 HOURS AFTER CHLORPICRIN GASSING.
EXTREME OEDEMA OF LUNGS; DILATION OF HEART

body is cold so that shivering occurs, the requirement for oxygen will be doubled. With hard work the oxygen requirement may be four times as much as at rest or more.

Figures giving the liters of air breathed per minute follow:

	Liters
Resting in bed	7.7
Standing at rest	10.4
Walking 2 miles per hour	18.6
Walking 3 miles per hour	24.8
Walking 4 miles per hour	37.3
Walking 4½ miles per hour	46.5
Walking 5 miles per hour	60.9
Very vigorous exercise	62.0 to 67.0[1]

These figures seem rather high. But Hill and Lupton found by the Douglas bag method on a subject running on a track, that oxygen consumption varied from 422 cc. per minute at rest, to 4175 cc. when running at a rate of 9 miles per hour.

The therapeutic value of complete rest and warmth in conserving oxygen is self evident. Often a gassed case may appear to be in good condition and will wish to walk in. Under no conditions should such a patient be evacuated otherwise than by stretcher and ambulance. Any gassed case must be regarded as serious from the start. All cases should be removed from the neighborhood of the poisonous gas at once, and must wear masks in the danger zone. Gases may cling to the clothing and envelop the patient in a poisonous cloud. In such cases the clothing should be changed as soon as possible.

Hot coffee is an excellent first aid treatment. It helps conserve heat, the caffeine is a good cardiac stimulant, and coffee also acts as a general bracer and relieves the weakness. First aid treatment may therefore be summarized as follows:

1. Remove from the gas.
2. Loosen clothing and equipment.
3. Keep quiet in recumbent position and keep warm. Evacuate on stretcher as soon as possible.

During the war, further treatment consisted chiefly of venesection and the administration of oxygen. English physiologists at first emphasized the administration of oxygen and were sceptical concerning the benefits of venesection. American authorities on the other hand, particularly Underhill and his collaborators insisted

[1] See page 26, Technical paper 82, Bureau of Mines.

on the great value of venesection and thought that oxygen was of minor importance. The fact is that both are equally important, and German, French, English and American observers finally agreed upon this point. Venesection should be practised before the development of extensive pulmonary edema. When thus used venesection leads, as animal experiments have shown, not only to a rapid dilution of the blood, but it maintains this dilution for a considerable time, so that the original concentration and the concurrent pulmonary edema never recurs. It exerts a powerful, beneficent influence on the entire course of the disease.

On the other hand, if pulmonary edema has already become extensive, and anoxemia is marked, as shown by cyanosis and a rising pulse rate, the administration of oxygen is imperative. Moreover oxygen is the remedy *par excellence* for the grey cases. Venesection should not be used in these cases where it only accentuates the collapse.

VENESECTION

Blood to the extent of 1 per cent of the body weight or 700 cc. may be withdrawn from men at the height of symptoms. If the case is seen early, satisfactory results may be attained by a smaller bleeding (0.5 per cent of body weight or about 350 cc.). The hemoglobin is then followed at one-half-hour intervals. So long as the hemoglobin does not increase, no further venesection is indicated. But if the hemoglobin rapidly increases, indicating that blood concentration is occurring again, a further venesection of from 350 to 500 cc. may be performed. The large majority of animals so treated survive without further treatment. It has been said that venesection is as necessary in the treatment of pulmonary edema as is the ligature for arterial hemorrhage. In advanced cases the blood may have become so thickened that it is difficult to remove over 50 cc. of blood. The administration of 0.25 gram caffeine, hot coffee, or camphorated oil may assist.

A cannula should not be inserted in the vein owing to the ease with which the blood coagulates. A simple incision should be made in a vein of the arm. Massage of the arm and active movements of the hand will accelerate the flow. Should the blood refuse to run, physiological saline may be infused into a vein in the other arm until the flow commences.

Intravenous injections of saline solution. These were highly recommended by Underhill on the basis of experiments on animals. Laqueur and Magnus found that intravenous saline caused a rapid diminution in the concentration of the blood, but that in comparison with venesection its use is counterindicated. The main value of venesection is in producing a fairly permanent dilution of the blood, and this extra fluid can only be derived from the tissues, particularly from the edema fluid. As the blood will become diluted without the salt solution, it is difficult to understand what the addition of salt solution will accomplish, except to prevent any further decrease in the edema fluid.

Since the war, the observations of all nationalities have become available including the very extensive experimental study of the treatment of phosgene poisoning by Laqueur and Magnus. In addition, experimental work on the treatment of phosgene poisoning has been carried out for several years by the Medical Research Division at Edgewood Arsenal. From the information so obtained, we may say that the treatment of phosgene poisoning should be carried out in accordance with the following logical program.

1. Counteracting the immediate effects of the gas (causal treatment).
2. To reduce or limit the amount of pulmonary edema.
3. To prevent the concentration of the blood.
4. To relieve the anoxemia.
5. To support the heart and circulation.
6. To relieve subjective distress.
7. To prevent infection and subsequent pneumonia.

1. *Counteracting the immediate effects of the gas.* The damage is done by the intense acid reaction produced by the phosgene in the pulmonary tissue. If this acid could be promptly neutralized, the damage would be greatly lessened. Inhalations of ammonia were used for this purpose by the English and Americans. But Laqueur and Magnus showed clearly on experimental animals not only that this treatment was of no avail, but that it actually increases the mortality, because it was a further source of irritation.

We have found that injections of urease, a ferment that acts on the urea of the blood liberating ammonia, exerts a beneficial action when it is injected intravenously immediately after gassing. Presumably the ammonia so produced is liberated directly into the tissues of the lung, and thus neutralizes promptly a certain portion of the acid.

The urease is prepared by making a 10 per cent water solution of jack bean meal. An active solution, when mixed with a solution of urea, will turn phenolphthalein pink in less than one minute. One cubic centimeter of this extract is injected intravenously for each 10 kilos of weight, so that about 7 cc. should be the dose for a man of average weight. The action of the urease may be hastened by a simultaneous injection of a few grains of urea. Dogs treated in this manner have developed pulmonary edema more slowly and lived longer than untreated control dogs gassed at the same time.

2. *To reduce the amount of pulmonary edema.* Fluid escapes into the lung because of the great dilatation of the pulmonary capillaries and the resultant stasis. If the pulmonary capillaries could be kept constricted, edema would not occur. It seems probable that the injection of urease effects this capillary constriction in addition to neutralizing part of the acid. The mucous membranes of animals so treated become blanched. However this action is fugacious. A number of drugs have been tested, but the only one that produces this effect over a long period of time is emetine. It has long been known that ipecac reduces pulmonary congestion, and emetine appears to have a constricting effect upon all capillaries, and has been used as a treatment for hemoptysis.

Moreover the action of emetine is prolonged, because its absorption is slow. In treating animals gassed with lethal doses of phosgene, we have found it advantageous to give a single full dose of emetine hydrochloride subcutaneously at the same time that the urease is given intravenously. Animals so treated have developed pulmonary edema much more slowly than untreated animals, or animals treated by urease alone. One-sixth grain emetine is used for each 10 kilos weight, or one grain for a man of average weight.

3. *Prevention of the concentration of the blood. Injection of gum glucose solution.* Attempts have been made to prevent the development of pulmonary edema by increasing the colloids of the blood to such an extent as to prevent the loss of fluid from the capillaries. Laqueur and Magnus found that the intravenous injection of a 25 per cent glucose solution saved the life of half of the animals gassed by phosgene that otherwise would have surely died. Barry found in 1923 that edema of the lung clears up to a varying extent when the colloid osmotic pressure of the blood is raised by a 10 per cent gum solution.

Experiments conducted at Edgewood Arsenal have shown that dogs may be safely given intravenous injections of a solution composed of 25 per cent gum arabic with 25 per cent glucose. Five cubic centimeters of this solution is injected slowly for each kilo of body weight. It was found in a series of animals, that if this injection is given soon after gassing (one to two hours), that animals receiving a lethal dose of phosgene may be saved by this treatment alone. This treatment is very much more efficient than venesection, and will actually prevent the development of pulmonary edema unless superlethal concentrations of phosgene are used. Gum glucose solutions may be prepared and sterilized in advance at field hospitals, and may be administered at casualty clearing stations, or possibly even at first aid stations, since the equipment required for such injections is simple in the extreme, consisting simply of a hypodermic needle of a fairly large bore, rubber tubing and a glass irrigator. The irrigator is filled with hot solution, the needle introduced into a vein, and the injection given slowly. It cannot be given very rapidly since the viscid solution runs through the needle with some difficulty. About twenty minutes should be used for the injection. When given more rapidly it causes some respiratory embarrassment and nausea.

To recapitulate, the treatment of pulmonary irritant gassing in the field should include:

(1) Intravenous injection of urease with or without urea and a subcutaneous injection of 1 grain emetine hydrochloride at the first aid station. This treatment prolongs the onset of pulmonary edema by several hours, which affords the patient a better chance in any case, and especially may afford the opportunity to transport him to the casualty clearing station or field hospital where other methods may be used.

(2) Injection of gum glucose solution at the earliest possible moment.

4. *Measures to relieve the anoxemia.* When superlethal concentrations are used, pulmonary edema will develop in spite of the use of gum glucose solution, but only after a greatly prolonged period. Thus a dog that has been treated with gum glucose solution may begin to develop pulmonary edema after twenty to twenty-four hours, while the untreated control may have died in eight to ten hours. For the treatment of the pulmonary edema and resultant anoxemia that occurs, oxygen is required. The result of oxygen therapy is marvellous. An animal that is gasping for breath and is deeply

cyanosed, breathes easily and the gums become pink within a few minutes after he is placed in an atmosphere of pure oxygen. The same results were obtained in the treatment of human cases. Even the grey cases resume a normal color. There are only a few cases in which the administration of oxygen fails to completely relieve the cyanosis. With the disappearance of cyanosis the results of anoxemia also vanish. The headache is relieved, the anxiety disappears, the breathing becomes quiet, the sensorium clears and the pulse improves. Patients become quiet and fall asleep with the mask on. If the oxygen is interrupted, the patient returns to his previous condition more or less rapidly, and in many cases he will himself ask for the mask to be replaced.

Oxygen should therefore be administered continuously and over a long period. The oxygen may be administered by the Haldane apparatus or some similar device which is provided with a mask fitting over the nose and mouth. Improvised methods of giving oxygen are seldom of any value. The best of these methods is administration through a nasal catheter. From 2 to 10 liters of oxygen per minute should be used, or whatever is required to keep the patient's face pink. There will always be need to conserve the supply of oxygen, and therefore it should not be wasted by giving more than is required to keep the patient in good condition. When the face becomes pink and the pulse slows down, he is receiving enough. This treatment must be maintained for hours or even for a day or two, with gradual reduction in the amount of oxygen until recovery is assured and the patient does not become cyanosed when the oxygen is interrupted.

Pure oxygen is irritating to the lungs if it is continued long enough, but it at least requires a very long exposure. Men have worn oxygen masks in mine rescue work for at least eight hours without injury. Animal experiments indicate that pure oxygen can be administered safely in a closed chamber for from twenty-four to thirty-six hours, and that 40 to 60 per cent oxygen can be breathed almost indefinitely without injury. It will only be necessary to administer pure oxygen during the peak of the edema. Probably no case will require pure oxygen more than twenty-four hours, a period that is entirely safe. As rapidly as possible the percentage of oxygen should be reduced to 60 per cent and then 40 per cent.

When breathing pure oxygen from a bag and exhaling into the room, there is some possibility that the carbon dioxide tension in the

blood may become reduced to such a point that the respiratory center will not be stimulated sufficiently, and respirations will not be efficient. This may be avoided by rebreathing into a bag, or by providing a mixture of oxygen with 1 to 2 per cent carbon dioxide. This should cause a very full ventilation of the lungs and assist in reducing the anoxemia. Such precautions are however unnecessary except when oxygen is administered continuously over a very long period. It may also be noted that oxygen cylinders should never be lubricated with anything except a very pure grade of glycerine. Several disastrous explosions have occurred when oil was used to lubricate valves of oxygen apparatus.

5. *Measures to support the heart and circulation.* With the development of obstruction to the circulation in the lungs there is a tendency toward dilatation and weakness of the right heart. Camphor and caffeine hypodermically act strongly on the heart and circulation and also on the central nervous system. These drugs are especially applicable before transportation. Alcohol is also a good stimulant, and a small amount of wine or brandy may be used for this purpose. American authorities have condemned the use of digitalis, but German authorities recommend it highly. It is useless given by mouth as it requires seventeen to twenty-two hours for absorption and action. Strophanthin 0.25 mgm., or digipurat, digitalen, or other of the digitalis preparations are recommended intravenously. As with venesection it is claimed that one should not wait until the high point of the disease is reached but that it should be used prophylactically while the heart is still good. When the pulse improves the digitalis should be discontinued. It may be repeated in four hours if required. Caffeine used intravenously is perhaps an even more reliable cardiac stimulant than digitalis, strophanthin or atropine even when these drugs are used intravenously. Two grains caffeine sodiobenzoate may be injected intravenously.

6. *Measures to relieve subjective distress.* The thirst from which patients suffer may be relieved by free administration of water, or better tea or coffee.

Cough is generally beneficial when it results in the expectoration of fluid. Violent and racking cough following chlorine gassing is often relieved when the patient is put to bed. It may be useful to give inhalations of various soothing mixtures to relieve this condition. Morphine should not be used. In case of necessity a little codeine may be used. The headache is a symptom of anoxemia, and is best treated by oxygen inhalations.

In a later stage, when bronchitis has developed, potassium iodide and ipecac have been highly recommended as expectorants.

Drugs that are counterindicated. There is very little drug treatment other than the above. Most remedies are injurious. The effect of morphine and phenacetin and similar drugs on the circulation is bad. Adrenalin has been tried and found injurious. The same may be said of pituitrin. Amyl nitrite is a heart tonic but it lowers blood pressure, which is already falling rapidly.

7. *Measures to prevent infection.* After the gassed cases have recovered from the acute pulmonary edema, they will need careful nursing and watching for several weeks. In gas poisoning we are dealing with a serious affection of the lungs, and there is always a possibility of infection and development of broncho-pneumonia. There should be a careful mouth hygiene, especially among those seriously ill, the air should be kept dust free as far as possible, and any cases of pneumonia should be promptly removed from the other gassed cases in order to prevent the transfer of infection. If laboratory facilities are available a routine swabbing of the throat of gassed cases and an examination for pneumococci and hemolytic streptococci, would be advisable. Cases harboring these organisms could be treated in separate rooms or wards, and thus the great majority of gassed cases might be protected from pneumonia, resulting from the introduction into the ward of a relatively small number of carriers of these organisms.

Prognosis. The future of an individual case can be told by the condition of the heart and the hemoglobin. A rising hemoglobin and red cell count indicates that the edema is progressing. A falling hemoglobin indicates that the peak of edema has been passed. If the heart stands up through the peak of the edema, the changes of recovery are excellent. The passage of a case from the blue type of asphyxia in which the face is congested and cyanosed to the gray type with an ashen pallor and pale lips indicates failing cardiac action and a correspondingly gloomy prognosis.

It must be remembered that cases that have been gassed with chlorine run their course to a fatal issue much more rapidly than cases gassed by phosgene, and that if treatment is to be successful in chlorine poisoning it must be correspondingly early.

It becomes a matter of acute interest to determine how long such cases should remain in hospital, and when they will be fit for duty again. A study of this question was made in England based upon

4575 gassed cases. In brief it was found that such cases averaged six weeks in hospital. But no such general rule will serve, some cases requiring less and others much more. A prolonged stay in hospital probably shortens eventual convalescence, and a short stay may mean a long subsequent convalescence. The surgeon must base his decision on the severeness of the primary gassing and symptoms immediately following, or the presence or absence of pulmonary complications and the actual condition of the patient at the time.

In this same study the total period of disability, i.e., the period between the date of gassing to the date of rejoining the Reserve Station, was studied. The duration of this period varied greatly. The majority were returned to duty between the tenth and the nineteenth weeks. But 25 per cent of the cases required over twenty-six weeks to return to duty.

Of these cases 67 were readmitted to hospital for symptoms referable to gas poisoning, and it was found that practically the same symptoms that prolong the original stay in hospital are operative in necessitating readmission. Of these 67 cases, 42 eventually returned to duty, while 6 or 9 per cent were discharged as permanently unfit. It will thus be seen that of the total 4475 cases only 6 or approximately a tenth of one per cent were finally discharged as unfit for further duty.

The question as to when a man is fit for duty is to be settled preferably by a practical test. The man should walk a measured mile briskly with an observer. If he cannot do this, or if this exercise produces dyspnea, or disordered heart action it is sufficiently obvious that he is not yet fit for duty. Should he pass this test, he may repeat this walk with his pack. By making practical tests of this character on cases that appear fit from a medical examination, there should be few cases returned to hospital.

There is a type of case, fortunately rather rare, in which convalescence is disturbed by the occurrence of attacks somewhat resembling spasmodic asthma. These attacks may occur every night, or less frequently. During the attack, the patient is obliged to sit up in bed, and his respiration is shallow and rapid, but not difficult, in which respect it differs from asthma. Slight cyanosis may be present, and the patient is anxious. Often the pulse is rapid during the attack, at other times slow and full. Such cases are suffering from polycythemia. The red corpuscles are increased to six or seven million, and the hemoglobin to 110 or higher, and this condition may

persist for several weeks. This condition was studied by Barcroft, Hunt and Dufton. These observers found that the symptoms complained of were: (1) disordered cardiac action; (2) nocturnal dyspnea; (3) high red blood counts; (4) low percentage of CO_2 in alveolar air; (5) acidosis and kindred conditions. The whole train of symptoms is the same as that exhibited at high altitudes.

The treatment consisted in keeping the patients in a chamber containing an atmosphere of 40 to 50 per cent oxygen. The CO_2 and aqueous vapor were removed by passing the air over soda lime and calcium chloride. As a rule the patients remained in the chamber for sixteen out of twenty-four hours for five days. They slept in the chamber every night, left the chamber after breakfast, and returned again just before tea. The tests used were: (1) a blood test (of polycythemia), (2) a pulse test, and (3) a respiration test.

These observers reached the following conclusions:

1. A description is given above of 26 cases of gas poisoning. They were treated by the continuous administration of oxygen. The patients were placed in chambers in which the percentage of oxygen aimed at, and usually maintained, was between 40 and 50. The usual term of residence was five days, sixteen or seventeen hours of each day being spent in the chambers. The night was always spent in them.

2. *Red cell count.* The effect upon the red cell count is as follows: If the red cell count is markedly higher than 5 millions, it is reduced by residence in oxygen, usually to just about that figure; if the red count, as is often the case, especially with mustard gas, is not raised above 5,000,000, residence in oxygen does not alter it. The subsequent history of cases differs: In some the red count tends to rise, in others it remains nearly normal at the end of two and one-half months. As the red count fell, there was usually a fall—but a much slighter one—in the hemoglobin. The colour index therefore rose.

3. *Pulse.* An almost constant sign in the gassed cases which we have studied has been a failure on the part of the pulse to return normally after exercise to its resting rate. This condition has been benefitted by the oxygen treatment in nearly all cases; and in about half it was brought to—or almost to—the normal condition.

4. *Nocturnal dyspnea.* Of fourteen cases treated in series I, six suffered from nocturnal attacks of dyspnea. None of these had attacks in the chamber; three appeared to be permanently cured, and two others permanently benefited.

5. *Breathlessness on exercise.* No measurements were made in the first series of cases; the patients, however, all stated that they were able to walk faster and with less distress than before the treatment. In the second series of cases a definite test for dyspnea was introduced, in which the opinion of the patients was not involved. Of the 6 cases thoroughly tested, 4 increased the quantity of work which they could do with a given degree of breathlessness by over 20 per cent, and 2 others by over 10 per cent.

A still smaller number of cases suffer from long continued dyspnea on exertion. Haldane believed that this is partly due to the fact that the alveolar epithelium has not entirely recovered, because the administration of oxygen enables them to perform muscular work more easily. But this is not the whole explanation because even with oxygen, the pulse remains abnormal and breathing is increased in frequency but not in depth as it should be. Haldane's explanation which appears to be reasonable, is that inspiration and expiration are governed by a reflex described by Hering and Breuer. This reflex fires off depending on the degree to which the center is stimulated by CO_2, and breathing naturally became deeper during muscular work. The dyspnea in chronic gas cases, like the "soldiers heart," is caused by neurasthenia. The central nervous system which ordinarily controls the Hering and Breuer reflex is exhausted, and the reflex becomes excessive. The patients cannot restrain it. This is shown by the fact that such patients cannot hold their breath for more than a few seconds. It may also be shown by breathing against a resistance. When the resistance is excessive, breathing becomes shallower and more frequent and anoxemia results, and face becomes blue. This change is due to exhaustion of the respiratory center. When this occurs, the reflex gets the upper hand and controls the breathing. In some persons this effect is produced more readily than in others.

In some cases of gas poisoning the respiratory center becomes exhausted and neurasthenic, and reflexes run riot through weakening of central control. Exhaustion in general is the cause of all neurasthenia. "Soldiers heart" is caused from overexertion. The treatment of such a condition, which is a very common basis for claims for pension or war relief, is in reality the treatment of neurasthenia. Gassing is only responsible for this condition in so far as it has led to the development of neurasthenia. In other words, it must be clearly recognized that the more or less permanent dyspnea and dis-

ordered heart action suffered by a small proportion of gassed cases, is functional, and is not caused by any organic disability suffered by the lungs themselves.

Hunt and Jones analyzed 320 chronic sufferers from irritant gas poisoning. Of these cases 116 were too slight to present a characteristic clinical picture. The remaining 204 were divided as follows:

A. *Cardiac cases* (116 or 56.3 per cent). They resembled the "irritable heart" cases. The characteristic symptoms of this group are precordial pain, dyspnea, exhaustion and persistent tachycardia after exercise. The exhaustion begins after a comparatively short period of exercise such as a half mile walk in ten minutes. The pulse is rapid and does not fall so quickly as in normal subjects.

B. *Spasmodic dyspnea* (37 or 18.1 per cent). The characteristic symptom is nocturnal spasmodic dyspnea, with increased cell count and hemoglobin.

C. *Mixed cardiac and dyspneic* (52 or 26.6 per cent).

Treatment. The cardiac cases improve if trained with graduated exercises. From the results of over 900 observations on the pulse rate, the following rule for prescribing the amount of exercise was established. On the fourth day after being allowed out of bed, the patient walks ½ mile in ten minutes. If the pulse rate has not fallen to 86 within five minutes after the end of the test, the exercise has been too severe. If the pulse rate has fallen to below 76 within five minutes, the exercise has not been excessive.

Oxygen treatment cannot be relied upon to improve the condition of patients suffering from this "effort syndrome." On the other hand, oxygen treatment as described by Barcroft, Hunt and Dufton is the best treatment for the cases suffering from spasmodic dyspnea.

The ultimate fate of gassed cases is discussed further in Chapter XV.

REFERENCES

(1) Gilchrist: Medical Aspects of Chemical Warfare, War Department, Chemical Warfare Medical Division, Washington, D. C.

(2) Official History of the War, vol. II, London, 1923.

(3) Hill: Lecture on gas poisoning. Jour. Royal Army Med. Corps., 1916, vol. 26.

(4) Klotz: Acute death from chlorine poisoning. Jour. Laboratory and Clinical Medicine, September, 1917, vol. II.

(5) Dunn: Tissue changes in animals produced by pulmonary irritant gases. Report No. 9 of the Chemical Warfare Medical Committee, London, September, 1918.

(6) Edkins and Tweedy: The histological effects produced by gas poisoning and their significance. Report No. 2 of the Chemical Warfare Medical Committee, London, April, 1918.
(7) Laqueur and Magnus: Experimentelle Pathologie der Phosgenvergiftung. Zeitschr. für die gesamte Experimentelle Medizin., 1921, XIII, 31.
(8) Schneider and Truesdell: The circulatory responses of man to a sudden and extreme anoxaemia. Amer. Jour. Physiol., July, 1923, V, 65, p 379.
(9) Meek and Eyster: Experiments on the pathological physiology of acute phosgene poisoning. Amer. Jour. Physiol., 1920, 51, 303.
(10) Barcroft: Anoxaemia as a factor in acute gas poisoning. Jour. Royal Army Med. Corps, 1921, vol. XXXVI.
(11) Douglas: Anoxaemia and its effects as seen in gas poisoning. Jour. Royal Army Med. Corps, 1921, XXXVII, 37, 216.
(12) Haldane: Lung irritant gas poisoning and its sequelae. Jour. Royal Army Med. Corps, 1919, XXXIII, 494.
(13) Haldane: Respiration. Yale University Press, 1922.
(14) Dunn: Report on necrosis of the cortex of the kidney in goats as the result of gas poisoning with phosgene and chlorpicrin. Report No. 9 of the Chemical Warfare Medical Committee, London, September, 1918.
(15) Hill and Lupton: Jour. Physiology, 1922, vol. 56, no. 5, p. 32.
(16) Underhill: The Lethal War Gases, Physiology and Experimental Treatment. Yale University Press, 1920.
(17) Laquer and Magnus: Experimentelle und theoretische Grundlagen zur Therapie der Phosgenerkrankung. Zeitschr. für die gesamte Experimentelle Medizin, 1921, XIII, p. 200.
(18) Barry: Pulmonary œdema and congestion in heart-lung preparations. Jour. Physiol., August, 1923, vol. 57, p. 368.
(19) Smith, Lorraine: The influence of pathological conditions on active absorption of oxygen by the lungs. Jour. Physiol., 1897–1898, vol. XXII, p. 315; also vol. XXIX, 1899, p. 19.
(20) Karsner: The pathological effects of atmospheres rich in oxygen. Jour. Exper. Med., 1916, vol. 23, p. 149.
(21) Haggard and Henderson: The treatment of carbon monoxide poisoning. Jour. Amer. Med. Assoc., October 1, 1921, vol. 77, pp. 1065–1067.
(22) The administration of oxygen in irritant gas poisoning. Report No. 10 of the Chemical Warfare Medical Committee, London, October, 1918.
(23) Polycythaemia after gas poisoning and the effect of oxygen administration in chambers in the treatment of chronic cases. Report No. 4 of the Chemical Warfare Medical Committee, London.
(24) Treatment of patients suffering from "effort syndrome" by continuous inhalation of oxygen. Report No. 12 of the Chemical Warfare Medical Committee, London.
(25) Hunt and Jones: The later effects of irritant gas poisoning. Report No. 15 of the Chemical Warfare Medical Committee, London.

(26) Report on the length of stay in hospital in the United Kingdom, and the disposal of gas casualties, Report No. 16 of the Chemical Warfare Medical Committee, London.

(27) Barcroft, Hunt and Dufton: The treatment of chronic cases of gas poisoning by continuous oxygen administration in chambers. Quarterly Journal of Medicine, 1919–1920, vol. 13, p. 179.

(28) Henderson and Paul: Oxygen mine rescue apparatus and physiological effect on users. Technical paper 82, Bureau of Mines.

CHAPTER VIII

The Vesicants

MUSTARD

Mustard was first used against the British at Ypres. The British called it mustard gas from its characteristic smell; the French named it Yperite after the locality where it was first used; and as the Germans used it in shell marked with a yellow cross, all parties frequently referred to it as "yellow cross." Its code designation H.S., is supposed to stand for Hunstoffe.

Physical and chemical properties. The compound called "mustard gas" is a heavy oily liquid. In the pure state it is colorless and nearly odorless, but in the impure state in which it was used during the war, it is dark colored, and in low concentrations has an odor strongly suggesting mustard or garlic. In high concentrations it is decidedly pungent and irritating.

Mustard gas is B B dichlordiethylsulphide (thio-diglycol-chloride) having the following formula:

$$(Cl \cdot C_2H_4)_2S \text{ or } \begin{matrix} ClC_2H_4 \\ \diagdown \\ \quad S \\ \diagup \\ ClC_2H_4 \end{matrix}$$

and must not be confused with the true natural mustard oil which is prepared from the seeds of black mustard and which is allylisothio cyanate, $CH_2{:}\,CH{:}CH_2N{:}CS$.

At ordinary temperatures mustard is a rather stable compound, but at high temperatures it decomposes, yielding hydrochloric acid and highly toxic and lacrymatory gases of unknown composition.

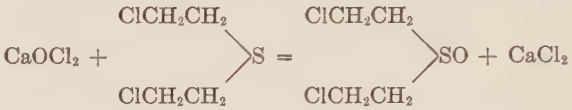
On contact with water mustard undergoes hydrolysis, slowly at ordinary temperatures and more rapidly on heating. In this reaction hydrochloric acid and dihydroxyethylsulphide $(HO{:}C_2H_4)_2S$ are formed as follows:

$$2HOH + (ClC_2H_4)_2S = (HO \cdot C_2H_4)_2S + 2HCl$$

Dihydroxyethylsulphide or thiodiglycol as it is often called is a non-toxic substance and hydrochloric acid in small concentrations is also

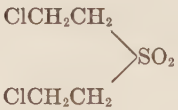
harmless. Thus mustard which has been scattered on the ground is eventually rendered harmless by hydrolysis. The rapidity of this action depends upon the amount of mustard in any one place, the amount of rain, and the temperature. But in general it is accomplished in from two days to two weeks or more.

Hypochlorites react violently with mustard, oxidizing it to mustard sulfoxide and evolving a great deal of heat. The reaction that takes place is as follows:



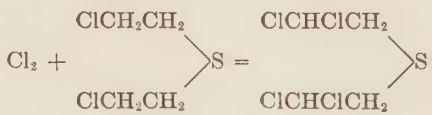
The sulphoxide is non-toxic, and for this reason calcium hypochlorite has been used to destroy mustard.

Curiously enough, when very strong oxidizing agents are used, two atoms of oxygen are added and the resulting product called mustard sulphone or



is very toxic.

Chlorine likewise attacks mustard vigorously, producing compounds having a higher content of chlorine which are harmless. Hence chlorine or chlorinating agents such as sulphurdichloride, SCl_2, and dichloramine T may also be used to destroy mustard.



From two to four chlorine atoms may be added in this manner, and none of these chlorinated mustard compounds are toxic.

Mustard is comparatively insoluble in water, but is readily soluble in hydrocarbons of the petroleum series such as gasoline and kerosene, and also in other organic solvents such as carbon tetrachloride, carbon disulphide, alcohol, acetone, or monochlorbenzene, C_6H_5Cl. Hence these solvents may be used to remove mustard from the clothing or the body. Mustard is readily dissolved in rubber, another hydrocarbon, and on this account readily penetrates rubber boots, gloves and other articles of clothing, especially if these are

brought into direct contact with liquid mustard. For this reason rubber articles do not afford protection against mustard for any extended period of time. Mustard proof clothing, which will be referred to later, should be used by workers in mustard or by details in destroying mustard.

Mustard melts at 13.9° when pure and boils at 219.5°, but at this temperature some decomposition takes place. The Specific Gravity of mustard at 0°C. (the solid) is 1.362.

The use of a material like Mustard, particularly where the vapor phase of the substance is relied upon to produce the desired result, depends chiefly on the vapor tension, because as the vapor tension is high or low, the maximum quantity that the air will contain is large or small. The vapor tension of course varies with the temperature. This is shown roughly in the following table:

TEMPERATURE		VAPOR PRESSURE IN MM. OF MERCURY	MAXIMUM CONCENTRATION PER LITER
Degrees Centigrade	Degrees Fahrenheit		
		mm.	*mgm.*
40	104	0.45	3.66
13.9	57	0.07	0.62
0	32	0.031	0.28
−17.8	0	0.0045	0.045

More accurate figures for vapor tension are available at temperatures usually encountered as follows:

TEMPERATURE		VAPOR PRESSURE IN MM. OF MERCURY	SATURATION POINT PER LITER
Degrees Centigrade	Degrees Fahrenheit		
		mm.	*mgm.*
15	59.0	0.0417	0.401
16	60.8	0.0457	0.439
17	62.6	0.0499	0.480
18	64.4	0.0546	0.525
19	66.2	0.0596	0.573
20	68.0	0.0650	0.625
21	69.8	0.0709	0.682
22	71.6	0.0773	0.743
23	73.4	0.0842	0.810
24	75.2	0.0916	0.881
25	77.0	0.0996	0.958
30	86.0	0.1500	1.443
35	95.0	0.222	2.135

The low vapor pressure of mustard explains why it vaporizes so slowly and is so persistent. The figures also explain why it is possible to pass through a mustard drenched area in cold weather with comparative safety, particularly in a high wind, when it would be dangerous to do so on a warm calm day.

Mustard is lethal in concentrations varying from 0.006 to 0.2 mgm. per liter, according to the length of the exposure. In general 0.07 mgm. per liter may be considered as lethal for thirty minutes exposures, and it is therefore approximately five times more toxic than phosgene, which as we have seen is ten times as toxic as chlorine. This great toxicity of mustard explains why it is so effective in spite of its low vapor pressure. For on a hot day it is theoretically possible to obtain a concentration of 3.66 mgm. per liter of air which is about fifty times the lethal dose, and even on a cool day (57°F.) the air will hold about ten times the lethal dose, and at freezing temperature, four times the lethal dose.

Of course these maximum concentrations can be attained by evaporation, only when the air remains for some time in contact with the liquid, i.e., when there is no wind. As there is always some wind in the field, it is not to be expected that these theoretical concentrations can be obtained under field conditions. However, if only a light wind is blowing, and this passes over a considerable area drenched with mustard, the concentration will approach the theoretical maximum and will become very dangerous. Moreover when mustard is dispersed in the form of a fine spray or mist by the explosion of a mustard shell, the vaporization is greatly hastened because of the greater surface of liquid exposed to the air, and higher concentrations will be obtained than can ordinarily occur from vaporization of the liquid from the ground. In fact when mustard is thoroughly atomized in this way it may remain for some time floating in the air as a mist, thereby yielding concentrations far above even the theoretical saturation of the air at that temperature. Moreover mustard is often mixed with solvents such as carbon tetrachloride, chlor benzene and carbon bisulphide, which have a lower freezing point and a higher vapor pressure. As a result the mustard is more readily dispersed both in cold and warm weather. It is possible that mustard could be used in this manner in aerial bombs, and would produce many casualties around the area of the burst. But when used in this manner the persistency of the mustard is correspondingly decreased.

Mustard was the vesicant gas of the last war. It was the most effective chemical agent used, and produced more casualties than any other gas. Thus of 36,965 gas casualties in which the kind of gas was stated, 27,711 were due to mustard. According to the report of the Surgeon General for 1919, mustard caused the loss of 1,286,784 days to the American Expeditionary Force out of a total of 12,545,442 days lost as the result of all battle injuries. According to British figures (Official History of the War, vol. 2, p. 308) 124,702 or 77.5 per cent of the 160,970 total gas casualties admitted to the casualty clearing stations, reappear in the records of hospitals on the lines of communication with a definite diagnosis of mustard poisoning. It may be assumed that 80 per cent of the total gas shell casualties during the mustard gas period were due to yellow cross shell (mustard), 10 per cent to blue cross shell (arsenicals) and 10 per cent to green cross (pulmonary irritants).

Mustard has many characteristics favoring its use. It is toxic in concentrations that cannot be detected by the sense of smell; it penetrates all ordinary clothing and affects all parts of the body with which it comes in contact; it is a very persistent gas and its action is delayed; it is difficult to protect a command that must pass through a mustard area, and if the area is entered unconsciously, as may often happen, even the mask may not be worn. For, especially when pure mustard is used the odor is not strong, and men without a keen sense of smell may not notice it. The sense of smell soon becomes dulled, and it may be erroneously supposed that the mustard gas has disappeared when it is no longer perceived. Thus, mustard not only produces many casualties, but breaks down morale. No one knows with certainty that they can eat, drink, sleep or even sit down in safety. After a more or less prolonged exposure to such alarms the nerves are exhausted and morale becomes lowered.

Pharmacological action of mustard. In order to become familiar with the effects of the absorption of dichlorethylsulphide into the system, dogs and other animals have been injected with the substance. The simplest method of introducing the substance is by subcutaneous or intramuscular injection of olive oil solutions. The effects observed on an animal from the injection of a lethal dose in olive oil, are, after a latent period: salivation, hyperexcitability and convulsions, diarrhea, slow and irregular heart which becomes rapid before death, muscular weakness, and finally coma and death.

The symptoms elicited from intravenous injections are similar to those observed from the injection of olive oil solutions. While the solution is being injected, and for some time afterwards, the animal shows no effects, whatever. A record of the blood pressure, pulse, and respiration fails to show any appreciable effect at this time.

Within ten to twenty minutes after injection, however, an increased salivation is noticed. This soon develops into a very free flow of rather mucinous saliva. The next symptom observed is usually a diarrhea which may be accompanied by vomiting. This diarrhea is present until the death of the animal, and a few hours after the injection the stools frequently contain blood. After injection the respiration becomes rapid and if anesthesia has not been used, the animal shows a distinct hyperexcitability. At this stage, he may be frightened by a slight movement of the hand or unexpected touch and the eye reflex may be obtained by touching almost any part of the face. The gait soon becomes unsteady, movements of the muscles are spasmodic but uncontrolled, and apparently accompanied by tetanic contractions of the muscles. The knee jerk may be elicited by a touch of the finger. The animal soon becomes unable to walk or even to stand, and the violent spasmodic movements increase to the stage of convulsions, with extension of the hind legs and arching of the neck and back. The pulse, which may have been somewhat slowed, soon becomes irregular. Palpation suggests that the heart is dropping a beat occasionally. The dropped beat becomes more and more frequent until finally the heart is beating at one-half its former rate. If the chest is opened it may be clearly seen that the ventricles are beating one to every two beats of the auricles. Later the rhythm may even become 1 to 3. Stimulation of the vagi slows this apparent hyperexcitability. The blood pressure very slowly falls, and a few hours before death, the heart resumes its normal rate. At this stage, or before, it is found that the heart can not be slowed or in any way affected by strong stimulation of the vagi, although the respiration is easily inhibited. Section of the vagi has no effect upon the heart rate except a slight slowing. Apparently the vagus endings are paralyzed. The heart becomes feeble, there is a great dilatation in the splanchnic area, a high venous pressure, and the arterial blood pressure is falling slowly. The convulsions have ceased, and the animal lies in a coma. Death comes quietly in less than twenty-four hours after the injection. It is probably due to the weakening of the heart and the great dilatation

of the vessels in the splanchnic area. Autopsy reveals a more or less intense congestion of the intestinal mucosa which may extend from the pylorus to the anus, and is frequently accompanied by hemorrhage into the lumen of the intestine. The condition suggests the excretion of the dichlorethylsulphide into the intestine. These effects upon the heart, the alimentary tract, and the central nervous system are quite characteristic and unmistakable.

Effects of inhalation of large doses of the vapor. Some of the effects which have been observed from the injection occur when a dog is poisoned by inhalation of the vapor. When a dog is poisoned by inhalation of a very large dose of the vapor, practically all the effects obtained by injection, salivation, vomiting, bloody diarrhea, hyperexcitability and convulsions, slow, irregular pulse, becoming rapid before death, and paralysis of the vagi have been observed. This suggests that in high concentrations, dichlorethylsulphide may be partially absorbed through the lungs and cause systemic effects. However, this action has not been proved.

Fate of mustard in the body. It is claimed that further evidence of the absorption of the sulphide through the lungs is furnished by the detection of one of the products of hydrolysis, dihydroxyethylsulphide in the urine of animals poisoned by dichlorethylsulphide by inhalation. This hydrolytic product can also be found in the urine after the injection of mustard gas. But dihydroxyethylsulphide also may be recovered from the urine after the application of 0.1 to 0.2 gram per kilo to the skin of dogs.

This product of hydrolysis is not responsible for the effects of mustard gas. The other product of hydrolysis is hydrochloric acid and is not a very toxic substance. Relatively large amounts can be injected intravenously without producing any marked effect. This is readily understood. When injected intravenously it is immediately neutralized by the buffer action of the blood. The blood does not become acid and the tissues are never really exposed to the acid. When strong solutions are placed on the skin or mucous surfaces, or injected into the tissues an irritating effect is noticed. Hydrochloric acid, however, injected in very large doses, does produce very definite effects upon the animal and can cause death. Both products of hydrolysis of mustard gas are very readily soluble in water and very sparingly soluble in organic solvents, or, in other words, have a low lipoid solubility or partition coefficient. It would be expected from this that they would not readily penetrate cells.

Theories of action. 1. Dichlorethylsulphide is very slightly soluble in water and freely soluble in organic solvents, that is, has a high lipoid solubility or partition coefficient. It would therefore, be expected to penetrate cells very readily. Its rapid powers of penetration are practically proven by its effects upon the skin. Having penetrated within the living cell, hydrolysis might occur. The liberation of free hydrochloric acid within the cell would produce serious effects and might account for the actions of dichlorethylsulphide. The mechanism of the action of dichlorethylsulphide according to this theory appears to be as follows:

a. Rapid penetration of the substance into the cell by virtue of its high lipoid solubility.

b. Hydrolysis by the water within the cell, to form hydrochloric acid and dihydroxyethylsulphide.

c. The destructive effects of hydrochloric acid upon some part of the mechanism of the cell.

This theory is very simple, and has been very generally accepted. It is probable that mustard does hydrolyze within the cell, at least to some extent. But it is absolutely not demonstrated that the very minute amount of hydrochloric acid so discharged within the cell would be toxic. The smallest droplet of 5 per cent solution of mustard on the skin will produce a blister, and the amount of hydrochloric acid that can be liberated from such a minute droplet of mustard is infinitesimal. The cells are not only alkaline in reaction normally, but they are capable of withdrawing buffer substances from the blood and lymph with which they are bathed. It is inconceivable that the cells would not be able to neutralize these infinitesimal quantities of hydrochloric acid, which moreover are not liberated immediately, but little by little as hydrolysis occurs.

Again, if we assume that cell penetration depends on lipoid solubility, then compounds similar to mustard of the same order of lipoid solubility and speed of hydrolysis should give comparable blistering results. This is not the case. Mustard or dichlorethylsulphide is a powerful vesicant. One of the tetra chlor di ethyl sulphids which has a higher lipoid solubility and only slightly slower speed of hydrolysis, is absolutely non-vesicant. Mustard sulphone on the other hand, has a lipoid solubility of only $\frac{1}{20}$ that of mustard and hydrolyzes only $\frac{1}{100}$ as fast, yet it is a powerful vesicant. Many other examples could be cited showing that there is absolutely no relation between lipoid solubility plus rate of hydrolysis, and vesicant properties.

2. *Chemical activity of the sulphur atom.* Many compounds are changed enormously by the addition of sulphur to the molecule. However, any change in mustard from a valency of two to a higher valency must mean the transition through a non-vesicant compound, mustard sulphoxide. Furthermore tests conducted in the laboratories of the Medical Research Division have shown that the excretion product of neither the sulphoxide (tetravalent) nor the sulphone (hexavalent) contains the bivalent sulphur atom while the excretion product from mustard does contain it. The end product of mustard metabolism has been isolated from the urine and crystalline phenol and thiophenol condensation products formed. No trace of these have been found after sulphone or sulphoxide injection. We may therefore state that there is no reduction of sulphur valency within the body nor is there any noticeable amount of oxidation, for 44 per cent of mustard injected has been recovered. This is a very high percentage when the loss in various metabolic processes is considered.

3. *The formation of an unsaturated compound.* The formation of an unsaturated compound was considered possible because of the action of mustard with alcoholic sodium hydroxide where hydrogen chloride is split out and an unsaturated compound formed. It is known that the vinyl group $(CH_2 = CH \text{ or } H-\overset{H}{\underset{|}{C}} = \overset{H}{\underset{|}{C}}-)$ has a marked influence on the toxicity of a compound. Isolation of the end products of metabolism was successfully carried out following injections of both mustard and di vinyl sulphide and these end products were identical with those secured using the pure compounds. There was absolutely no change from one to the other. This shows that the toxicity of mustard does not depend upon the formation of a vinyl radicle, and no other toxic unsaturated radicle can probably be formed from mustard.

4. *Action of the entire molecule.* The belief that the toxicity is due to the action of the molecule as a whole was finally reached. In this connection it is interesting to note that British investigators have recently published some data which lead them to the same conclusion. Our investigations were more varied then theirs, for we have found that the action on enzymes is specific as is also the case with simple proteins. Yeast, for instance, is killed by mustard, while it is affected by neither hydrochloric acid nor di hydroxy ethyl sulphide, the two products of mustard hydrolysis. The action of

rennet, on the other hand, is accelerated in about equal amounts by all three of these compounds.

This theory is in accord with what we know concerning the action of toxic substances generally. The action of strychnine on the motor cells of the cord, is due to the entire molecule, and cannot be produced by any one of the radicles that may be split off from that alkaloid. Many of the alkaloids are very closely related in chemical constitution, yet their pharmacological action is very different. Each peculiar aggregation of molecules constituting the alkaloid appears to act as an entity, and we cannot say in any case that their pharmacological action depends upon any one of their contained radicles.

Whatever the theory of action, mustard is a cell poison, exerting its necrotizing action on all cells with which it comes directly in contact, including the skin and mucous membranes, with all their structures. The capillaries and other organs that mustard reaches become paralyzed.

It is well known that the injuries produced by mustard heal much more slowly than burns of similar intensity produced by physical or other chemical agencies. This characteristic is explained by this action of mustard on the blood vessels which are rendered incapable of carrying out their functions of repair; and by the fact that necrotic tissue acts as a good culture medium. Hence the great liability to infection of mustard burns.

Pathology of mustard gas poisoning. Gross pathology in acute cases (those dying within forty-eight hours). The skin lesions in acute cases vary greatly in their distribution, depending upon the severity of the exposure and the resistance of the individual. If a mask has been worn the face is generally clear. Lesions do not develop on the scalp. Any portion of the rest of the body, from the neck down to the shoe or boot top, may be the seat of a burn. There is often a normal belted area around the waist where protection has been afforded from the action of the gas by a tight fitting belt. The nature of the lesions in the early cases varies between slight and extreme grades of erythema and slight and extreme degrees of vesicle formation. In severe cases all of these lesions may be found in the same individual on various parts of the body. The parts earliest affected are the axillae, bends of the ebows, inner aspects of the arms and thighs, groins and genitals.

The *eyes* show either a marked catarrhal conjunctivitis or a more advanced purulent conjunctivitis. There is no alteration in the cornea in early cases.

In the nose, the pharynx and on the posterior third of the tongue the mucous membrane is edematous, and occasional patches of necrosis are found.

On opening the thorax the *lungs* do not collapse. They show depressed dark areas of atelectasis, alternating with elevated light pink areas of emphysema, and also some normal areas. On section the cut surface is moderately dry but the vessels are greatly engorged with blood. The large bronchi and trachea are lined by a diphtheritic false membrane which forms a complete cast of these passages, and which often extends to the smallest bronchi. A number of the bronchi are found plugged with exudate and it is possible that this may account for the atelectatic lobes.

The heart shows no change except some dilatation of the right ventricle.

All the abdominal organs show marked passive congestion. Occasionally necrosis and even ulceration of the mucosa of the stomach is found, and this is thought to be due to the eating of food contaminated by mustard gas. A hemorrhagic condition of the mucosa of the stomach and small intestine is also sometimes found.

Microscopic findings. Skin sections from various regions show necrosis of the epithelium, in some places the necrotic process extending for various depths into the corium and occasionally going entirely through the corium into the subcutaneous tissues.

The lungs. There is an enormous engorgement of the blood vessels with some edema. There is considerable atelectasis with compensatory emphysema. In many cases there is practical disintegration of the lung parenchyma. The parenchyma appears as a mass of engorged capillaries, desquamated epithelial cells, round cells, a few polymorphnuclear leucocytes and red cells. This under low power has the appearance of a true inflammatory pneumonia. Under high power, however, very little inflammatory reaction is seen. The polymorphnuclear leucocytes are not abundant and there is little or no fibrin present. The picture is that of the "chemical pneumonia" of some authors.

In the upper respiratory passages there is found destruction of the epithelium with often necrosis of the entire mucous membrane. A false membrane is constantly encountered and this is composed of

fibrin, leucocytes, red cells, epithelial cells, mucus and debris. There is necrosis of the walls of the bronchioles and of those of surrounding alveoli with hemorrhage and early inflammatory exudate. Bacterial stains show bacteria in the false membrane and in the walls of the small bronchioles, Gram-positive cocci predominating.

Gross pathology in later deaths (those dying after forty-eight hours). The *skin lesions* in these cases present a great variety of conditions. The areas which were originally only erythema and did not go on to vesicle formation, now show a dark brownish pigmentation of the skin with dry desquamation of the outer layers of the epidermis. Vesicles are found in various stages of drying, some full of fluid, others completely dried up. Denuded and necrotic areas are also found, in which places there is considerable secondary infection. The worst lesions are found in the parts earliest affected, namely, the genitals, axillae, bends of the elbows and inner aspects of the arms and thighs.

The *eyes* may show no lesions, having entirely cleared up before death, which is generally due to secondary infection elsewhere. There may still remain, however, a purulent conjunctivitis, and occasionally a corneal ulcer is encountered. Lesions more severe than this are seldom encountered in men as the eyes are easily treated from the earliest stages. Much severer lesions are encountered in experimental animals.

The nose, pharynx and posterior third of the tongue show far more extensive necrosis than is present in the acute cases, and sometimes false membrane formation is found. There is a marked purulent inflammatory reaction around the necrotic areas.

The *lungs* are the seat of an extensive necrotizing broncho-pneumonia with abscess formation. Bacterial cultures can always be obtained from these lungs and it is evident that the pneumonia is due to secondary infection and is the principal cause of death in these cases. The bronchi and trachea contain a thick purulent exudate in addition to the false membrane which is found in various stages of disintegration. The lumen of the small bronchi is entirely occluded with pus and on section they appear as abscesses.

The *heart* shows no lesion due to the specific action of the gas. The right ventricle is dilated due to the obstruction of the pulmonary circulation.

There is passive congestion of all abdominal organs.

Specimen from Fatal Case of Mustard Poisoning, Showing False Membrane on Tongue and in Trachea

Similar Case Showing Necrotic Mucosa, Sloughing and Ulceration

Microscopic findings. The *skin* shows various stages of necrosis depending upon the severity of the lesions in the region from which it is taken. There is also some inflammatory reaction not encountered in acute cases. Some fibroblastic proliferation is encountered in the corium or subcutaneous tissues at the lower border of the necrotic areas. Regeneration of the epithelium is also found beginning from the hair follicles and sweat glands.

The respiratory passages. Very similar lesions are present in the various parts of the respiratory tract from the naso-pharynx to the bronchi. The lesion may be described as an acute membranous inflammation with associated vascular and cellular changes.

The earliest change appears to be a simple vascular engorgement. Capillaries, venules and veins are all involved, and in some cases is extreme, and is sometimes associated with hemorrhagic extravasation into the cellular tissues.

Capillary and venous thrombosis ensue, especially in the superficial, sub-epithelial zone. The smaller vessels are blocked by dense plugs of fibrin, and in the larger vessels there is a more open fibrinous network.

Epithelial disintegration and desquamation and the formation of an inflammatory membrane occur simultaneously. The membrane is composed of fibrinous exudation forming a network in whose meshes are leucocytes, epithelial cells and large numbers of bacteria. This membrane may extend to the smaller bronchi, and the bronchioles beyond may contain varying quantities of fluid pus. The epithelium in the bronchioles may be intact, but more often is infiltrated with leucocytes and is ruptured, permitting free communication with the pus in the bronchiole and that in the surrounding pneumonic patch.

The inflammatory membrane is associated with a variable degree of fibrinous inflammation in the living subjacent mucosa and submucosa accompanied by leucocytic infiltration both focal and diffuse, and edematous infiltration.

The lungs. Vascular engorgement, edema, and catarrhal inflammation of the alveoli are present in practically all cases, usually with inter-alveolar hemorrhage, suppurative broncho-pneumonia, and fibrinous pneumonia. Vascular engorgement is the most uniformly constant change. The capillaries and venules of the alveolar walls, and the larger vessels are equally involved. Edema is seen most frequently in relation to patches of broncho-pneumonic consolidation,

where it occupies the intervening lung tissue. Generally the edematous portions of lung are also intensely engorged and varying degrees of edema, catarrhal desquamation, leucocytic infiltration and intra-alveolar hemorrhage are seen. The intra-alveolar hemorrhage is an accompaniment of the congestion. In milder cases it is due to diapedesis of red cells, but in the more severe cases, actual hemorrhagic infarction occurs and the alveoli are stuffed with recent blood, and the alveolar walls show more or less necrosis. Areas of interstitial hemorrhage also occur and there is considerable hemorrhage into the alveolar walls.

Varying degrees of emphysema and atelectasis are combined with these changes. Emphysema is not a conspicuous feature. A few emphysematous patches are found here and there. Small areas of collapse are usually seen in relation with the patches of broncho-pneumonia.

Summary. The lesions that cause death are due to an acute inflammation of the entire respiratory tract, followed by desquamation of the mucous lining and formation of a diphtheritic membrane. This membrane plugs the air passages and affords lodgement for bacteria. Acute purulent bronchitis and broncho-pneumonia follow infection.

Symptoms of mustard gas poisoning. An extraordinary set of symptoms is produced by mustard, yet they are readily understood and classified. Whatever its mode of action may be, it is known that mustard acts—first as a cell irritant and finally as a cell poison. It kills any cells in the body with which it comes in contact, and all of the symptoms produced may be explained as the result of this poisonous action upon the cells. Apparently it requires a certain time for this damage to the cells to be produced, for the symptoms do not appear immediately after exposure. Only in rare cases, where the worst forms of poisoning result from the direct application of liquid mustard, do symptoms appear during the first hour. In most cases the first symptoms appear in from four to six hours, but a latent period up to twenty-four hours may occur. The length of this latent period depends chiefly upon the concentration of the gas to which the victim has been exposed, and the higher the concentration the shorter the interval between exposure and first symptoms as a rule. This rule has some prognostic value for it may be expected that when symptoms appear shortly after exposure, the case will probably be severe and vice versa. There is nothing absolute

about this because the latent period and intensity of symptoms also depend largely upon the individual's susceptibility to the poison. There is a great latitude among people in this respect, some individuals being quite resistant, others very susceptible, with all grades between.

The symptoms produced by mustard are local and general as follows:

I. Local. 1. Conjunctivitis or inflammation of the eyes.

2. Erythema of various skin areas, which may be followed by blistering and excoriation or ulceration.

3. If the mask has not been worn, inflammation of the nose, throat, trachea, and bronchi, with possibly necrosis and sloughing of the mucous membrane, followed by the development of secondary bronchitis or pneumonia.

4. If any of the gas has been swallowed there will be a similar inflammation of the mucous lining of the gastro-intestinal system, resulting in nausea, vomiting and diarrhoea.

II. General. Gastro-intestinal symptoms, effects on the nervous system such as profound apathy and depression, effects on the circulation, and disordered metabolism associated with loss of appetite.

History of a typical unprotected case. On exposure to the vapor or to a finely atomized spray of mustard, nothing is noticed at first except the faint though characteristic smell. After the lapse of several hours, usually four to six, the first symptoms appear. The systemic symptoms are intellectual dullness or stupidity, headache, oppression in the region of the stomach, nausea or vomiting, malaise, and great languor and exhaustion. In many cases these symptoms may not be noticed, and the local symptoms first attract attention. The eyes begin to smart and water. There is a feeling of pressure or often of a foreign body, and photophobia, and when examined the conjunctiva is found to be reddened. The nose also runs with thin mucus as from a severe cold in the head, and sneezing is frequent. The throat feels dry and burning, the voice becomes hoarse, and a dry harsh cough develops. Inflammation of the skin now shows itself as a dusky red erythema of the face and neck which look as though they had been sunburned, but are almost painless. The inner surfaces of the thighs, the genitals, the buttocks, the armpits, and other covered portions of the body are similarly affected. Mustard affects more severely those parts of the body where the skin is tender and well supplied with sweat glands. Itching and burning

of the skin may be spontaneous, or first noticed as the result of washing. Even these mild symptoms may be sufficiently irritating to cause sleeplessness. At the end of twenty-four hours a typical appearance is presented. The conjunctivitis has steadily increased in intensity, the vessels are deeply injected, and one of the main items of distress is caused by the pain in the eyes which may be very intense. The patient lies virtually blinded, with tears oozing from between bulging edematous eyelids, over his reddened and slightly blistered face, while there is a constant nasal discharge, and continuous harsh, hoarse coughing. Frontal headache is often associated with pain in the eyes and photophobia with blepharospasm is always marked. During the second day the burned areas of the skin generally develop into vesicles, and the scrotum and penis and other badly burned areas become swollen, edematous and painful to the touch. Bronchitis now sets in with abundant expectoration of muco-pus, in which there may later be found large actual sloughs from the inflamed tracheal lining. The temperature, pulse rate and respiration rate are all increased.

These symptoms now increase in intensity for several days if the case has been severely burned. On the other hand cases that have been only slightly poisoned may never proceed to the blister stage. The Clinical cases may therefore be divided into three groups:

1. *The mildest.* Objective signs are few, erythema of the skin, a reddening of the eyes, or a slightly hoarse voice. This may have been due to long exposure to low concentration. and after a few days these cases may develop severe general reaction and involvement of the lungs which may as result of infection end in death. Although this is not the rule, one cannot count on it, and it is only after three days observation that one can be sure they are mild cases.

2. Cases that either at once or at end of several days show extensive lesions of skin or respiratory tract, and eyes. Larger surfaces of the skin are affected, and there may be not only redness, but further changes. A hot distressing cough, aphonia, loss of appetite with severe pain in the intestinal tract and obstinate constipation. Eyes are swollen, dripping tears and there is severe blepharospasm. Are very sick, apathetic, and hide from light under bed clothes. The nose drips, and cough is continuous. There is fever and disturbance of circulation.

3. In the third group are the worst cases. With the picture of the most severe intoxication, very rarely without widespread local lesions. The picture is a serious disturbance of the central nervous system, widespread and deep changes in the skin, and mucous membranes, and severe ulceration and stenosis of the bronchial tree up to the alveoli. Fever is marked, and the circulation impaired. It is the picture of the second class increased in severity and complications are more frequent. These complications include influenza and infection with streptococci, pneumococci, and staphylococci. Some appear in twenty-four hours, but most infections only after several days, and the longer the illness lasts, the more probable are these intercurrent infections. On the skin they take the form of infected ulcers; in the respiratory tract they result in lesions varying from an infectious bronchitis up to the worst pneumonia. In such cases the intoxication of mustard is combined with bacterial intoxication, leading to the picture of severe sepsis. Death may rarely occur on the second or third day, more often up to the third or fourth week in the more lingering cases.

The eyes. The inflammation of the eyes appears as one of the earliest symptoms and varies from the mildest conjunctivitis with lachrymation and slight irritation to very severe injuries. However when a case is first seen it is impossible to say whether it will remain mild or whether it is only the beginning stage of a severe inflammation. Usually the latter occurs. The swollen closed eyes drip tears and pus. If the eye is opened, it will be found that the vessels are dilated, and the cornea may be involved. A severe keratitis with clouding occurs in a certain number of cases. Great care must be taken that the patient himself does not make the condition worse by rubbing the eyes, and proper treatment initiated to prevent ulceration of the cornea. Fortunately this is rare and occurs mostly in cases in which liquid mustard was splashed in the eyes. Usually and in uncomplicated cases, after several days the swelling and photophobia decrease markedly, and the clouding of the cornea also clears up after several weeks. Blindness rarely results. Secondary infection is most to be feared, and is to be prevented by avoidance of rubbing by the patient and careful washing and treatment by the attendants.

British authorities classified eye injuries into three groups.

1. The milder cases, 75 per cent of the total who become fit for duty in from one to four weeks.

2. Intermediate cases, 15 per cent of total, who recovered in from four to six weeks.

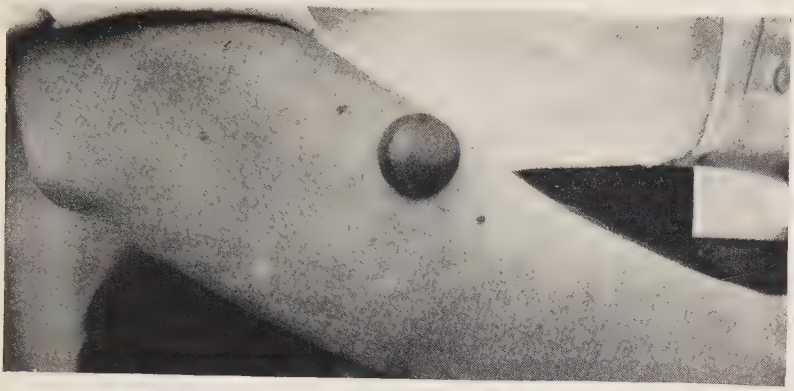
3. Several cases with corneal changes, 10 per cent of total, who required from two to four months for recovery.

The skin. When the action of mustard is limited to the effect of the vapor alone, the affection is localized to uncovered or slightly protected regions, and to areas where the skin is tender or thickly set with glands, such as the face, neck, the armpits, groins, genitals, thighs, buttocks. Places on the trunk where the clothes are fastened tightly as under the belt or straps of the pack. Parts of the body where the skin is thick such as the scalp, the palms of the hands and soles of the feet are seldom affected.

The affected area is reddened as by sunburn or by the action of a mustard plaster. There is always more or less deep and well marked infiltration of the dermis. In places where the subcutaneous tissue is loose, as in the eyelids or the scrotum, edema is more clearly defined, the skin being smooth and bloated. These lesions produce subjective symptoms of burning, itching and tension.

From the first complaints about burning and itching, and from cases having only the slightest lesions which resemble sunburn closely, the lesions run through the different degrees of burning with vesicle formation to destruction of tissue with ulceration. Some few cases are uniformly burned over the entire area of the body. Such bright red swollen patients may present a picture of severe systemic disturbance, even though the lesions do not proceed to vesiculation. The fever, intestinal disturbance, and nervous symptoms may be due partly to absorption of mustard, and partly to absorption of products of decomposition from the skin. The course of such cases is for the most part unfavorable.

After the second day, if blisters do not form, the deep red erythema gives place to a copperish, lavender or brownish color which finally becomes a brown or black pigmentation. When this pigmentation occurs it persists for several weeks, or until complete exfoliation of the epidermis. The degree of this pigmentation apparently bears little relation to the severity of the burn. It is often very annoying when occurring on exposed parts of the body, especially the face. However it disappears in all cases eventually. This pigmentation reminds us strongly of the similar color resulting after sunburn, and may have a similar causation.



EXPERIMENTAL MUSTARD BURN, 24 HOURS AFTER APPLICATION

MUSTARD BURN, 24 HOURS AFTER CONTACT

SAME MUSTARD BURN, FOUR DAYS AFTER CONTACT

When blistering occurs numerous small vesicles usually appear first which gradually coalesce into large blebs. The blebs contain a clear yellow serum, and if the epidermis is removed, a raw surface is left. There is little pain unless the skin becomes infected. Blisters may continue to develop for several days in crops. This has led to the belief that the blister fluid contains mustard which may cause reinfection if allowed to flow over the skin. This seems highly improbable, and the successive development of lesions is rather to be explained by the various reaction to different intensities of exposure by areas of varying sensitivity.

The respiratory tract. Although the changes in the eyes and skin produce more subjective and objective symptoms, the damage to the mucous membrane of the respiratory tract is the most serious part of mustard poisoning. The mucous membrane of all air passages is affected, from the nose, pharynx, larynx, trachea and bronchi, down to the alveoli of the lungs. As elsewhere, the rapidity with which symptoms appear, the intensity and extent of the injury depend chiefly upon the concentration of the gas, the time of exposure and the susceptibility of the patient. The respiratory tract may be affected in persons not protected by a mask, even though the skin has been burned very slightly. The lesions vary from the slightest catarrhal inflammation to the formation of a diphtheritic membrane that may fill the entire bronchial tree. The first symptoms are dryness and tickling of the throat and a raw feeling behind the sternum. This is followed by a discharge from all mucous surfaces as may be seen in influenza. The hoarse voice and the barking cough of these patients is quite distinctive. On inspection, the mucous membranes are all reddened, and covered first with mucus, later with muco-pus.

Laryngitis may be intense with complete aphonia, and frequent, painful cough. Local examination shows the existence of edema predominating in the region of the epiglottis, and paralysis of the vocal cords. Expectoration becomes abundant and muco-purulent, and may contain large flakes of mucus and sometimes false membrane, which rarely may even cause obstruction of the larynx.

If the lungs are examined during this earlier stage nothing is found save some alteration of the breath sounds, possibly some vesicular râles. Later the entire chest is filled with râles, as in any case of bronchitis. Increasing cough and rising temperature and pulse indicate the development of severe bronchitis.

If the affection proceeds to the depth of the alveoli, or if the inflammation of the bronchial mucous membrane proceeds to necrosis, membrane formation, and hemorrhages as this membrane separates, the systemic symptoms, fever, rapid pulse are accentuated and in addition cyanosis adds its train of symptoms.

If infection is added, various degrees of broncho-pneumonia, abscess formation, and involvement of the pleura develop with their accustomed symptoms and physical signs, and the condition of the patient becomes critical. The expectoration is enormous and may amount to as much as a liter in twenty-four hours, and consists chiefly of pus mixed with blood and shreds of membrane.

Acute broncho-pneumonia is the most serious and most frequent complication of mustard casualties. It occurs, from the third to the eighth day of the illness. It may be ushered in by chills, headache and general malaise, but the invariable preliminary symptom is an elevation of temperature, with rapid pulse. Dyspnoea may be considerable. This broncho-pneumonia may cause death in several days. On the other hand the pulmonary infection may persist in a prolonged type of broncho-pneumonia which in time produces marked cachexia and death in several months.

Certain patients make a prompt recovery. The pulmonary phenomena disappear progressively, the general health improves, the fever disappears, and a return to duty seems near, when suddenly the broncho-pneumonia reappears.

The prognosis in such cases is bad, yet many of the processes clear up, and patients make a good recovery after several months of chronic bronchitis.

The gastro-intestinal tract. In nearly every case there are certain symptoms indicating involvement of the gastro-intestinal tract. These consist of pressure and pain in the epigastrium, nausea and vomiting, and loss of appetite. Occasionally there may be diarrhea but as a general rule there is constipation lasting several days. These phenomena may be explained as the local action of mustard that has been swallowed with the saliva. In some cases food or water that has been contaminated with mustard has been consumed, and in such cases the gastro-intestinal symptoms are most marked, and may occur alone without skin or lung involvement. In such cases there will also be pain in the small intestine or colonic region, and the loss of appetite is very marked, together with great general depression. In general in these cases it may be said that when the appetite begins to pick up, improvement has set in.

General intoxication. Since mustard is a substance that is absorbed into the tissues unchanged, there is always a possibility that a certain amount may be absorbed into the circulation, especially in very severe and extensive poisoning, and thereby set up a toxic action upon the entire organism. It is difficult to say with certainty whether mustard is actually thus absorbed unchanged but many symptoms have been explained by some observers on this basis. Some of the gastro-intestinal symptoms may be explainable in this way. The effect of mustard upon the circulation, the nervous system and the general metabolism has been thus explained by many observers.

Injection of mustard into dogs results in an increased excretion of total nitrogen, ammonia, creatinin, and phosphoric acid, indicating an increased destruction of tissue, the exact cause of which is not understood. This corresponds with the clinical observation that many cases of mustard poisoning suffer from great depression, loss of appetite, cachexia, and loss of weight that may last for several months before recovery. It is sometimes difficult to decide how much of this picture is due to intoxication and how much to psychical disturbances. In certain cases the continued loss or gain in weight may aid in resolving this doubt.

The alterations in metabolism may also be partly caused by the action of mustard upon the various glands, especially the liver, pancreas, and intestinal glands. That the function of these glands will be disturbed if mustard is absorbed is to be expected. But no permanent injury to these glands is found at necropsy. The urine in most cases is likewise negative, though occasionally albumen, blood and casts may be found.

The blood. Stewart made a study of the leucocytic changes in cases seriously gassed with mustard including six cases that subsequently died.

From the first to the third day after gassing, there is usually a great increase in the total leucocyte count which is due to a large increase of polymorphonuclear neutrophils. Thus in one case on the second day after gassing, the total leucocyte count was 35,000 of which 33,800 (96.6 per cent) were polymorphonuclear neutrophils. At the same time there is a moderate or great reduction in the number of lymphocytes, while the eosinophil and basiphil cells practically disappear.

As the disease progresses toward a fatal issue, and in the very severe non-fatal cases, about the third or fourth day, there is a rapid fall in the polymorphonuclear count, reaching in the last day or two an excessively low figure. Thus in one case the neutrophils fell from 33,800 on the second day to 15,800 on the third and to 172 on the seventh, six hours before death.

These leucocytic changes may be of some prognostic value. Stewart felt justified in drawing the following tentative conclusions:

1. A very high relative neutrophilia, 90 per cent or upwards, with or without an increased total leucocyte count, indicates a bad prognosis. Of 7 cases in which the neutrophil polymorphs were present in proportions varying between 92 and 99 per cent, 6 died, and the seventh was very seriously ill for some days. On the other hand a series of seven slight and moderately severe cases showed an average of 60 per cent neutrophils, the highest figure being 68 per cent.

2. A bad prognosis is also indicated by a rapid fall in the total leucocyte count to an extremely low figure, this usually taking place between the third and sixth days.

3. Maintenance of a good eosinophil count or the appearance of an eosinophilia, relative and absolute, is a good prognostic indication, great reduction or total absence of these cells, a bad indication.

4. In the present series of cases the leucocytic changes found merely confirmed an already fairly obvious prognosis, good or bad. It is possible that in other cases these changes may have real prognostic value, either in warning the physician that a case apparently slight or doing well may yet be a proper source of anxiety, or in showing that a patient whose outlook seems bad may still have elements of hope in his case. Further work is necessary to determine these points.

Considering the great severity of many of these cases when first seen and the long period of convalescence required by many, it is surprising how few sequelae or permanent disabilities remain. This may be because all of the very serious cases die. It is safe to say, however, that after a year little objective evidence of having been gassed can be demonstrated. Eye lesions are of the rarest. Unless the skin lesions have resulted in ulceration with great destruction of tissue there is no trace of the action of mustard on the skin. A few cases of chronic bronchitis with bronchiectasis or emphysema may be found, but in the majority of cases there are no pulmonary symptoms a year after gassing, although the x-ray may show the shadows caused by peribronchitis. This subject is discussed in more detail in Chapter XV.

It must also be remembered that the really serious cases accumulate in hospitals and it is these cases that have been chiefly described

in the literature. A much fairer idea of the results of mustard gassing can be obtained from following a considerable number of gassed cases with tabulation of their symptoms. This was done by Wilson and Mackintosh, who analyzed a consecutive series of 1500 mustard gas casualties admitted to No. 7 Stationary hospital between January 1 and October 31, 1918. During this period mustard gas was responsible for 77 per cent of all gas casualties as shown by the following table:

Gas cases of all kinds admitted May–August (inclusive), 1918

	MAY–JULY		AUGUST		TOTAL	
	Number	Per cent	Number	Per cent	Number	Per cent
1. "Mustard" gas	254	77	438	77	692	77
2. "Blue cross"	35	11	50	10	85	10
3. "Phosgene"	17	5	34	6	51	6
4. "Lachrymatory"	13	4	25	4	38	4
5. "Not defined" British, etc.	9	3	19	3	28	3
Total admissions	328	100	566	100	894	100

The incidence of the commoner functional manifestations in this series of 1500 mustard casualties is shown below:

FUNCTIONAL DISORDER	NUMBER OF CASES	PERCENTAGES
Functional photophobia	189	12.6
Functional aphonia	108	7.2
Functional vomiting	16	1.0
D. A. H.	19	1.2

All degrees of laryngitis were encountered from the slight case with hoarseness and painful cough to a grave condition associated with the formation of a false membrane.

The table following represents the relative frequency of the laryngeal complications.

Total number of cases, 1500

	NUMBER OF CASES	PERCENTAGES
Slight laryngitis	115	7.6
Severe laryngitis with complete or partial aphonia	81	5.4
Definite ulceration of vocal cords	22	1.4
Functional aphonia	108	7.2

The next table shows the proportion of all mustard gas cases admitted during four months, in which were noted (*a*) a definite pyrexia (apart from trifling initial reaction), (*b*) bronchitis and broncho-pneumonia, and (*c*) definite burns (apart from slight erythemas and scorching).

Total number of mustard gas cases, 692

	NUMBER OF CASES	PERCENTAGE	TOTAL
Pyrexia:			
a. Definite pyrexia, but mild in type, and not sufficient to cause long retention	181	26	37.4
b. Severe, causing delay in hospital beyond a month, as from severe burns, broncho-pneumonia, etc.	78	11	
Bronchitis, etc.:			
a. Milder types, not causing retention in hospital beyond a month	71	10	24.2
b. Severe bronchitis, broncho-pneumonia causing prolonged retention in hospital, evacuation to England, or death	97	14	
Burns:			
a. Lesions not severe enough to cause retention in hospital beyond a month	116	17	31.0
b. Severe, causing prolonged retention in hospital, evacuation to England, or death	98	14	

Effects on the eyes. There were two groups:

	Number of cases
1. Slight conjunctivitis requiring only about fourteen days' treatment in hospital	767
2. Severe lesions	352
Total number of mustard casualties in which eyes were affected (74.6 of all cases)	1119

The lesions in the second group were of three degrees of severity:

	Number of cases
1. Severe conjunctivitis with photophobia and lachrymation	253
2. Acute purulent conjunctivitis without corneal injury	69
3. Acute purulent conjunctivitis with corneal injury	30

Effort syndrome. In the whole 1500 cases, 125 men failed at first to respond normally. At the end of a short course of treatment

the great majority gave a normal response. In 19 men the symptoms of effort syndrome persisted in spite of graduated exercise. In fourteen of those men the symptoms were due to other conditions or had existed before the war, and only 5 were directly due to gas.

Intercurrent disease. In 4.5 per cent (75 cases) the original symptoms were overshadowed by new disease—influenza, common cold and diphtheria.

Period of invalidism. Invalidism may be considered under two groups.

1. Men left in France, 559 cases, 1 per cent died and 2 per cent were evacuated, to London. In 97 per cent of these cases therefore we ascertained the period that elapsed before each individual could march half a mile with his equipment. Eighty-seven per cent of all admissions passed this test within a month of gassing (57.5 per cent within fourteen days and 15.5 per cent within twenty-one days).

2. Men sent to England. Thirteen per cent of all mustard casualties. In these the period of invalidism lasted from three to six months.

Prophylaxis, sensitivity. Anyone who observes the tremendous differences between individuals is struck with the practical use that might be made of this difference. There are individuals who show distinct burns (erythema) from a fifty-second exposure, while working beside them are men who require an exposure of five to ten minutes to produce a burn. The hyperemia produced by the five seconds' exposure is not serious when limited to a small area, where it is closely surrounded by normal tissue, but a hyperemia of no greater intensity produced over a large area of the body may make it imperative that this individual be relieved from duty. The edema and itching may be quite distressing, and prevent the patient from sleeping or resting. He may have to spend most of his time bathing the injured parts with cold water to get relief. The man beside him, however, may be exposed to the same concentration for ten to fifty times as long, and show absolutely no effect. The former individual should not be allowed to work in any region where there is even a low concentration of mustard gas, for if he does, he is certain to be burned severely, and practically certain to become a casualty. The latter individual, on the other hand, would make a splendid workman in a mustard gas factory or a splendid soldier in a territory which has been shelled with mustard gas, if provided with a mask. Moreover, there is good reason to believe that the individual who is

sensitive to the skin effects will be sensitive to the eye and lung effects.

The objection has been raised that it is impractical to withdraw large numbers of men from organized military units. This is not a serious objection, for the following reasons: The sensitive individual will have to be withdrawn sooner or later as the result of injuries received. The advantages of removing him before he becomes a casualty are obvious. Moreover the percentage of sensitive individuals is small; by the proposed method it is not over 2 per cent. On the other hand the very resistant individuals are numerous, about 25 per cent. Further, it is not necessary that sensitive individuals be withdrawn until troops enter the zone of the advance where mustard may be encountered. Sensitive men should then be assigned to duties on the lines of communication.

Method of making the test. Three solutions of mustard are made in absolute alcohol: 0.01, 0.1 and 1 per cent. These solutions must be freshly perpared as hydrolysis of the mustard takes place on standing. The forearm of the man to be tested is bared. With glass rods which are drawn out to a fine point, a minute droplet of the 0.01 per cent solution, is placed on the flexor surface of the forearm near the bend of the elbow. With a second rod a minute droplet of the 0.1 per cent solution is placed near the middle of the forearm.

The arm is examined twenty-four and forty-eight hours afterward. If both droplets have produced a definite area of redness, the man is very sensitive. In the normal individual, the droplet of 0.01 per cent solution should cause no inflammation, and 0.1 per cent should produce a definite area of erythema. If neither of these solutions cause redness, the man may be tested with a droplet of 1 per cent solution. If this fails to redden the skin the man is very resistant. It has been stated that 78 per cent of negroes are resistant.

Hair retains mustard, and should be worn quite short in the field for this as well as for other reasons. The proper use of the mask will prevent the effects of mustard on the eyes and respiratory tract, and clothing can now be provided which will to a great extent protect the skin. These prophylactic measures are discussed at greater length in the chapter on individual protection.

Protective ointments. Most of these are totally useless, and all have a very limited value. Ointments have several disadvantages as follows:

1. We cannot cover the whole body.
2. They rub off.

3. Only a thick, impervious and unpleasant coating will protect.
4. They have oily bases which in some cases actually absorb mustard and increase the burn.

However, ointments may have some value, especially if they could be made to protect parts that cannot be covered by protective clothing such as the face and neck. Hence many attempts have been made to develop an ointment that will actually protect. The following ointment has been recommended for field use:

	Per cent
Zinc oxide	45
Linseed oil	30
Lard	10
Neutral wool fat	15

This ointment was found to be of considerable value as a protective agent in mustard factories and shell-filling plants, but gave better protection against high or medium concentrations than to a long exposure to low concentrations. It should only be used on those portions of the body that are not covered with protective clothing. Protective clothing is far superior to ointments, and will undoubtedly be furnished to all who are liable to be exposed to mustard.

First aid treatment. The removal of mustard from the skin. It is generally recognized that the first step in the protection against and the treatment of mustard gas burns is to remove, as completely as possible, the mustard gas from the skin. The importance of doing this quickly does not seem to be sufficiently appreciated. The substance penetrates the skin with remarkable rapidity and unless the washing is done within a few minutes, a serious burn will result. In some cases, a perceptible hyperemia may develop even if the skin is cleansed a few seconds after contact. But as long as the odor of mustard gas can be detected on the skin, it is worth while to clean the exposed portion with a good solvent.

Soap. It was at once recommended that after exposure to mustard gas, men should bathe in hot soap solution. Later degassing units made provision for bathing large numbers of men, and undoubtedly greatly reduced the number of casualties in this way. The practice was also followed in many factories, with a large degree of protection to the men. However, it was found experimentally that washing with green soap solution five minutes after application of liquid mustard does not prevent or lessen the degree of the burn unless unabsorbed mustard remains on the skin at that time. It was further

determined that the addition of varying amounts of alkali, glycerol, alcohol, acetone, kerosene or gasoline did not increase the efficacy of the soap solution so long as these additions were not present in considerable concentration. Tincture of green soap was found to be the most effective soap because of its emulsifying ability.

Organic solvents. Kerosene, acetone, or absolute alcohol are effective solvents for cleansing the skin. Petrolatum, olive oil and lubricating oils are also satisfactory. The best ordinary cheap solvents are kerosene and gasoline, of which the less volatile kerosene is preferable. Pure mustard gas may be applied to the skin and if the area is quickly sponged with kerosene and afterwards washed with soap and water, only a faint hyperemia of no consequence will develop. On the dog's skin which is somewhat thicker, the burn was prevented by sponging with kerosene one minute after application of mustard gas (0.001 cc. per square centimeter of skin). Sponging the skin within ten minutes was distinctly beneficial but after fifteen minutes the treatment had little or no effect.

Further investigation has shown that in addition to the beneficial effect of washing the skin with kerosene, a marked benefit may be obtained by rubbing with kerosene for as long as thirty minutes, with frequent removal of the contaminated kerosene by a clean cloth. It has been shown repeatedly that of two burns, one of which was just sponged in the usual way with kerosene, followed by soap and water, and the other rubbed for some time with kerosene, the one which was not rubbed may blister, while the one which was rubbed does not. In this way, it is possible to prevent blistering as long as twenty minutes after pure mustard gas has been applied to the skin. This is impossible if the area is simply sponged. When soap and water is used in the same way, although the benefit is much greater than that which follows mere washing, it is far less effective than kerosene. It was found impossible to prevent blistering ten minutes after application of pure mustard gas to the skin, by the use of soap, whereas a similar area treated in the same way with kerosene showed only a faint hyperemia. The optimum length of time for rubbing appears to be about thirty minutes. The treatment is also quite effective against burns made by the vapors.

Chloride of lime destroys mustard by oxidation and chlorinating agents destroy mustard by further chlorination. It is an interesting fact that dichlorethyl sulphide is much more toxic and vesicant than monochlorethyl sulphide, but that the further addition of chlorine to dichlorethyl sulphide renders it quite inert.

In view of these facts which have been proven experimentally the following methods are suggested for the removal of mustard gas from the skin:

1. As quickly as possible, the exposed area is sponged with clean kerosene. If kerosene is not available, gasoline from an automobile may be used if due care is exerted to prevent fire. Alcohol may also be used if available. The kerosene or other organic solvent is then wiped off with a clean cloth, and more uncontaminated kerosene is applied. The area is then rubbed thoroughly with the hand. Every minute or two the kerosene is removed from the skin and the hand by a clean cloth. If all mustard is not removed by this repeated cleansing, the burn may merely be spread. The rubbing and removal of contaminated kerosene should be continued for about thirty minutes, and the parts are then thoroughly washed with hot water and tincture of green soap.

2. Should these solvents not be available, the following method may be used. Wash the affected parts as promptly as possible with an aqueous solution of chloride of lime or with Dakins solution. Dry chloride of lime should not be used as considerable heat is generated when the mustard is oxidized. After this has been thoroughly applied, wash well with hot water and tincture of green soap.

All of these agents for treating mustard burns should be kept at first aid stations or degassing units at the front. Casualties may be reduced to a minimum by the methods outlined.

Treatment of mustard casualties. It is presupposed here, that the prophylactic measures recommended were not used, or were used too late, and that casualties have occurred. The clothes of such men should be removed in an outer chamber and the men should be given a shower before admission to a ward. If this precaution is not taken, the mustard adhering to clothes and hair will be given off in the warm ward, and all patients there will be subjected to further irritation of these vapors.

The eyes. The object of treatment is to relieve irritation and to keep free from accumulated secretion, and prevent infection. The conjunctival sac should be thoroughly flushed, using a 1 per cent solution of sodium bicarbonate, or a saturated solution of boric acid or simple physiological saline solution. Use a syringe or douche cup, opening the eyelids wide, everting if possible. This treatment should be administered every two or three hours. In the milder

cases the only troublesome symptom is the photophobia which usually results, and the only treatment for this is protection against the light by proper eye shades. Should these not be on hand they may be improvised of brown paper.

In severe cases, pain may be severe and the use of cocaine is naturally suggested. However, all authorities state that the use of this drug is dangerous in that it tends to loosen the corneal epithelium. As damage to the cornea is to be faced in severe cases nothing should be done to increase this danger. Above all, the eyes should not be tightly bandaged or heavily compressed, and special care must be taken to prevent glueing of the lids and accumulation of secretion. The instillation of a few drops of sterile liquid petrolatum may prevent this glueing of the eyes. Warthin and Weller recommend highly the use of dichloramine-T 0.5 to 1 per cent in chlorcosane, as not only preventing the eyes from becoming sealed, but also preventing infection which is the main danger. In cases where the cornea has become affected and especially if ulcer develops, atropine should be used to dilate the pupil.

As soon as the acute stage of the inflammation has subsided the eye shades should be removed. A functional photophobia is one of the commonest and most troublesome sequels of mustard lesions of the eyes. In the treatment of these cases, the physician should express no anxiety concerning the outcome, but should inspire confidence that vision will be as good as ever in a few days. This is absolutely correct in the vast majority of the cases.

Treatment of the external mucous membranes. The nasal and pharyngeal mucous membranes should be thoroughly cleansed with bicarbonate solution. If done immediately after exposure it has a prophylactic value in removing mustard. Later it is all the treatment required. Laryngeal inflammation is best treated by direct applications or by sprays and inhalations. Menthol has been highly recommended for its soothing effect. Ten grains of menthol to 30 cc. of tincture benzoin compound may be used. Four cubic centimeters are added to boiling water, and the vapor is inhaled. Oil of eucalyptus has also been recommended for inhalations. In serious cases the French used *huile gomenolee.* This was warmed and injected into the larynx daily by means of a curved laryngeal syringe. This oil was likewise used when the trachea was blocked with sloughs thus producing dyspnea. In such cases the oil was injected by a fine needle through the crico-thyroid membrane.

The lungs. Mustard does not produce pulmonary edema, and bleeding is therefore futile and injurious. The effect of mustard per se on the lungs is not fatal except in relatively enormous dosage. The main danger lies in the greatly increased susceptibility to infection. Treatment should aim to reduce infection to a minimum. For this reason fresh and uninfected cases should not be treated in the same ward with pneumonias or other infected cases. Cases that have been exposed to mustard are probably as susceptible to streptococcus infection as a puerperal case and for the same reason. A very large area of mucous membrane is injured and partly devitalized. The wearing of a respiratory mask has been recommended and was actually used during the war with apparent good effect in reducing infections. The mask should be made of aluminum or other suitable metal, perforated and made to fit over the mouth and nose. It is retained in place by tapes. In the mask is placed absorbent cotton partially saturated with the following solution:

Rx.
Menthol.....2.5 grams
Chloroform.....8 cc.
Oil or eucalyptus.....8 cc.
Creosote.....8 cc.
Tinc. iodine.....4 cc.
Sp. Vini Recti q.s.ad.....60 cc.
Sig. 15 drops on cotton of mask every hour

A mask so applied should act as a filter for infecting organisms. Menthol, chloroform, eucalyptus, and creosote should all have a soothing effect on the mucous membranes and assist in preventing infection. The value of the iodine is problematical.

If broncho-pneumonia should develop, its treatment is the same as in other cases of pneumonia, namely symptomatic, with special effort to maintain good heart action.

The skin. The main purpose of treatment should be to keep the skin clean and prevent the infections that so greatly prolong healing. Ointments are as a rule counter-indicated both because they seal in infection and because their fatty bases are good solvents for mustard and prolong its action. Ambrine as used on heat burns has been extensively used for mustard burns and must be condemned, as infection proceeds apace beneath this impervious covering. Warthin and Weller recommend the following method:

A. *Mild injuries.* Wash immediately, or better, immerse parts one-half to two hours in Dakin's solution (strength about 0.5 per cent hypochlorous

acid). If too irritating, and when the skin is unbroken this is not likely to be the case, dilute the solution or shorten the time of immersion. If injured surfaces are very large, use full bath; for lesions of genital region, use the sitz bath. When immersion of parts is not expedient use wet Dakin's dressings or irrigate with Dakin's solution. If the wet treatment cannot be carried out, the application of dichloramine-T in chlorcosane or chloramine-T in sodium stearate may be used. These procedures serve a double purpose of chlorinating and rendering inactive any dichlorethyl sulphide remaining in the skin, and hair follicles, and also disinfecting the damaged skin surface. After the Dakin's solution use wet dressings, irrigation or bath of sterile hypertomic saline (5 to 10 per cent), alternating with Dakin's solution and sterile physiological saline according to judgment. As long as lesion remains an erythema, continue this treatment during the day time. At night, dry the skin under aseptic precautions, and apply a vanishing surgical cream containing hypochlorous acid of the type of chloramine-T, 1 per cent in sodium stearate surgical cream. Dusting powders are inadvisable because of crusting and possibility of infection. When vesicles form, empty vesicles early, under aseptic precautions, by means of a hypodermic syringe or sterile needle, with slight pressure upon the vesicle, and allow intact vesicle to collapse and seal down upon its base. After this procedure continue alternating Dakin's and sterile salines as above. If vesicles are not drained early, the fluid content may within 4 to 5 hours coagulate to such an extent that it cannot be removed, and becomes a possible medium for bacterial growth. It is very important that skin damaged by mustard gas be protected from trauma. Slight injuries, even ordinary pressures of the body, will cause the development of secondary vesicles or decubitus in the injured areas. In the case of denuded surfaces with much pain and when bedsores have developed, the colloidol saline bath is strongly advocated. This bath is made by dissolving 1 pound of commercial cornstarch and 1 pound of sodium bicarbonate in 20 to 30 gallons of sterile physiological salt solution, at a temperature of 90° to 95°. The patient can be left immersed in this, when necessary, from fifteen minutes to forty-eight hours. The patient should be constantly watched by the nurse. Should the pulse become weak, give strychnine or digitalis or remove from bath and apply blankets and heat. When severe lesions are upon the shoulders, buttocks, and back, the greatest care must be taken to avoid bedsores as they will develop very quickly. For this purpose the constant colloidol bath or saline bath, with patient lying in canvas body cradle to take off pressure, should be used.

The above method of treatment appears to be quite ideal except for the fact that when many casualties occur the military nursing personnel will never be sufficient to carry it out. If, however, instead of using irrigations or baths of Dakin's solution on all cases, a more or less permanent dressing of dichloramine-T in chlorcosane, or the 1 per cent chloramine in sodium stearate be used on all the less serious cases, results will probably be quite as good. I have seen these dressings used in a fair number of cases with good results.

Such dressings need not be renewed oftener than each 6–8 hours. When secondary infection does not occur an uncomplicated recovery may be expected within seven to ten days. The hands of the operator should be surgically clean in all cases.

Benzyl alcohol gives a great deal of relief from the itching that is so troublesome following mustard burns. It may be used as a lotion as follows:

Benzyl alcohol	100
Ethyl alcohol	96
Glycerine	4

Gastro-intestinal symptoms. Regulation of the diet is of the utmost importance. During the first few days when nausea and vomiting are frequent, the diet should be liquid. Milk and broth, if possible, but if these are not retained, a considerable amount of water should be given to aid elimination. The internal administration of 20 grains of sodium bicarbonate has been thought to be beneficial. Full diet should be returned to as soon as possible.

Management of the convalescent. To quote:[1]

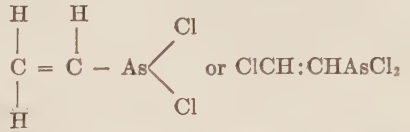
> The management of the convalescent period provides the great test for the medical officer's ability, because he is required not only to treat the disease, but to restore moral, to cut short hospitalization, and to lift men out of the slough of self-analysis which so often follows gassing. The best results will therefore be obtained by placing gassed cases in selected hospitals under the care of medical officers with special aptitude for this.
>
> As soon as a man is convalescent and free from the danger of septic complications, he should be discharged from hospital to a canvalescent centre, where a well-ordered routine of exercise, employment, amusement, and rest will quickly restore him to a state of physical and mental fitness.

LEWISITE

The compound called lewisite is so named after Professor Lewis who first isolated and described it in 1918. It had been prepared in impure form as early as 1904, but was not further investigated at that time because of its highly irritating and poisonous properties. It is the most important of the war gases developed by the American Chemical Warfare Service during the war, but has never been manufactured in large quantities and was not used during the war.

[1] Official History of the War, Medical Services, Diseases of the War, vol. II, p. 458, 1923, London.

Lewisite is a heavy, oily liquid and in the pure state it is colorless, but darkens on standing. Its vapors while extremely irritating have but very little odor which has been described as resembling geraniums. Lewisite is B chlorvinyldichlorarsine, having the following formula



It may be prepared by passing acetylene (C_2H_2) through arsenic trichloride ($AsCl_3$) in the presence of aluminum chloride $AlCl_3$ which acts as a catalyst. When the resulting oily liquid is distilled, it contains arsenic trichloride, lewisite, and also a mixture of two related compounds, B B′ dichlordivinylchlorarsine or $(ClCH:CH)_2AsCl$ and B B′ B″ trichlortrivinyl arsine or $(ClCH:CH)_3As$.

All of these compounds are liquids at ordinary temperatures having boiling points ranging between 190° and 260°C., and are all irritating and poisonous compounds.

Lewisite is the most active of all. It is soluble in oils, benzene (C_6H_6) and in the ordinary organic solvents. It is insoluble in water and dilute acids, but in contact with water lewisite hydrolyzes with great rapidity yielding its oxide ClCHCHAsO + 2HCl. This action is hastened by elevated temperature and the presence of alkalies.

Lewisite freezes at −13°C. and boils at 190°C. Its specific gravity is 1.920 at 0°C. and 1.8855 at 20°C. Its vapor pressure is comparatively low, 0.087 at 0°C. and 0.395 at 20°C. Thus we find that 1 gram molecule (207.3) would occupy $22.4 \times \frac{313}{273} \times \frac{760}{1.467} =$ 13,290 liters. Hence 1 liter of air saturated with lewisite vapors at 40°C. would contain 0.0156 gram or 15.6 mgm. per liter. If the air were saturated with this vapor at the freezing temperature, it would contain about 1 mgm. per liter.

As the lethal point for lewisite may be given as 0.048 mgm. per liter, and the blistering concentration at 0.334 mgm. per liter it would appear that this should be a most effective war gas. However it has never been tried in the field, and there is some doubt that this theoretical concentration could ever be maintained, owing to the

rapid hydrolysis of lewisite by water or aqueous vapor. In the laboratory lewisite is a powerful vesicant being quite equal or superior to mustard in this respect. It is also a powerful respiratory irritant.

Symptoms produced by lewisite vapors. When dogs are exposed to the vapors of lewisite, the first symptoms to appear are those resulting from irritation of the exposed mucous membranes. This is followed by disturbances of the alimentary tract leading to retching, vomiting, and excessive salivation. Following exposure the symptoms are the result of the severe lesions produced on the exposed mucous surfaces, including the lower respiratory tract. There is an inflammation of the entire tract down to the smaller bronchi, with a general congestion of the lungs and some edema. At this stage infection readily sets in and broncho-pneumonia frequently results, terminating usually in death.

During exposure, irritation of eyes, nose and throat commences at once. Blinking of the eyes is the first symptom in exposed animals, followed by excessive nasal secretion, lachrymation, retching and vomiting. If the exposure is continued these symptoms increase in severity, and dogs suffer excessive salivation and a marked inflammation of the eyes.

Following exposure, the animals are inactive with labored respiration. The nostrils are clogged with a thick membrane. Coughing is frequent, accompanied by ejection of a vomitus of frothy mucus probably swallowed. Many dogs die in this condition within two days after gassing. Should they survive this period the symptoms increase in intensity. There is frequent, violent sneezing, a watery or muco-purulent discharge from the eyes, and a nasal discharge so excessive as to cause ropes of mucus to hang, or a clear watery fluid to run continually from the nostrils. Coarse râles are general over the chest and indicate an intense bronchitis. Should the membrane which fills the respiratory passages begin to clear up, respiration becomes less labored, but in the majority of cases the symptoms continue unabated for from three to five days, during which time a few more animals die. Survival for this time is a favorable sign that recovery will occur. The membrane in the nose usually disappears by the fifth day, after which the main symptoms are coughing and conjunctivitis. Recovery is practically complete after from seven to ten days.

Temperature. During the first hour after exposure, all dogs show a drop of about one-half a degree in temperature. This drop is

followed by a rise to normal or slightly above. If death occurs within fourteen hours the temperature fluctuates around normal until just before death when it drops. In animals that live longer the temperature is subnormal during the first day. A rise is observed in the second day, but this is followed by a slow decline until death. This is true even for animals that have well advanced bronchopneumonia at death. The temperature therefore is no index to the severity of the effects of gassing.

Pulse. Practically all dogs showed a decreased pulse rate immediately after gassing followed by a return to normal or above before the expiration of the first day after gassing. During the second day the pulse was moderately increased, but after the second day the pulse rate was nearly normal.

Respiration. A rise in the rate of respiration immediately after gassing was usual. This rise persisted for the first day and tended to return to normal on the second day. A decreased rate of respiration was noted for some hours before death.

Pathology. Group I. Acute deaths. Dogs dying within sixty hours after exposure.

External examination of dogs dying within sixty hours after exposure to approximately a lethal dose of lewisite revealed a conjunctivitis with usually a watery discharge in the corners of the eyes. This discharge in the majority of cases was so profuse as to mat the hair of the entire face. Even as early as twelve hours a thick mucoid material completely plugged the nostrils. In the mouth there was usually a stringy or frothy material. In no case was any skin irritation noted.

On opening the neck the tissues around the trachea were usually intensely edematous. When the chest wall was removed it was seen that edema extended well down into the thoracic cavity involving, in many cases, not only the tissues of mediastinal space but those of the pericardial sac as well. This edematous condition was particularly characteristic of dogs dying within the first fourteen hours after exposure, but was seen in practically all deaths up to sixty hours. In all cases observed, the thoracic cavity was free from fluids and adhesions. The pleural surfaces were frequently injected and the lungs usually completely filled the cavity. Frequently the margins of the lobe met in the center and almost hid the heart. The lungs were heavy and of a rubbery or doughy consistency. The upper lobes were light or grayish red in color with many small purplish

areas. The red color represented the emphysematous areas, while the purplish color indicated congestion. Generally the posterior lobes were very dark red to purple in color, and were congested everywhere. All lobes were more or less edematous, but the edema was particularly marked at the hilum. Frequently small areas of consolidation could be felt, especially in the posterior lobes. In other cases, even when death occurred within twenty-four hours after exposure, pneumonia was so extensive as to involve all lobes. On cutting through such lungs the cut surface was seen to be dark red and meaty in appearance; the bronchial mucosa stood out prominently, and from the cut bronchi, fluid and sometimes pus issued. In some cases a false membrane could be pulled from the larger bronchi. This membrane was a continuation of the membrane which lined the nostrils, larynx, and trachea and which seemed to form a cast of the entire respiratory tree. Upon opening the larynx and trachea a thick grayish yellow membrane, thicker anteriorly than posteriorly and which rolled up when cut, was found closely applied to the wall. As it stripped from the wall the underlying surface was seen to be congested and edematous. If dogs survived the first twenty-four hours it was then seen that the membrane had started to break down and in some cases the lumen of the trachea was filled with a gray necrotic mass which tended to accumulate in the lower part of the trachea so that, at the bifurcation, the lumen was almost completely occluded.

Microscopic examination of a piece of trachea covered with this false membrane showed the tracheal mucosa to be destroyed and the submucosa congested and sometimes edematous. The membrane was composed of cell débris, desquamated epithelial cells, fibrin, mucus, leucocytes, and a few red blood cells. Examination of the lung tissue showed purulent bronchitis, broncho pneumonia, edema, congestion, hemorrhage, acute emphysema, atelectasis, and a widespread perivascular and peribronchial edema. In many cases the pneumonia was seen to be mixed with the edematous fluid giving a picture of pneumonia very different from the usual one. In some cases it was confined to the aveoli immediately around the bronchi, in others it was very extensive and involved many anatomical units.

A great many dogs which died acutely showed a marked dilatation of the right ventricle of the heart. Subpericardial hemorrhages were frequently found scattered over the surface, especially just below the coronary sulcus. In a few cases there were scattered sub-

endocardial hemorrhages. There was also a marked injection of all coronary vessels. Usually, however, the valves were normal. The pericardium, too, showed a characteristic condition. In many cases it was edematous, and in practically all cases its vessels were intensely injected. Excess fluid, however, was not found. The condition of the kidneys was very characteristic in the dogs dying acutely. They were dark bluish purple in color; the capsules stripped easily. Internally all zones were intensely congested and the boundary zone appeared violet red in color. The liver was congested but to not so great a degree as the kidneys. Other organs were occasionally hyperemic but usually were normal.

Group II. Later deaths. Dogs dying between three and nine days after exposure.

External examination of these dogs showed them to have a well developed purulent conjunctivitis. When death occurred during the fourth day after exposure a remnant of the false membrane was still found in the nostrils. In three dogs pleural effusions were found; in another, ascites was noted. Three dogs showed a membrane formation in the trachea; in another, the membrane had broken up and was simply a purulent mass; in the rest the laryngeal and tracheal mucosa was either just about normal or only slightly injected. Fifty per cent of these dogs showed broncho pneumonia with or without a purulent bronchitis. All dogs showed some type of bronchitis. The majority of dogs had lungs showing amounts of atelectasis varying from small scattered marginal areas to areas so extensive as to hide all lung structure. Congestion, edema, emphysema, and hemorrhage were also found in these dogs. The other organs of the body showed, as lesions referable to gassing, only a mild congestion of the liver and the kidneys.

Summary. A thick diphtheritic membrane is found in the nostrils, larynx and trachea, with a purulent bronchitis, and varying degrees of congestion, edema, hemorrhage, atelectasis and emphysema of the lungs. Broncho pneumonia is usually found. There is also an intense congestion of the liver and kidneys in animals dying acutely. The average time of death was thirty hours. Of 29 dogs studied as types of acute death, 23 died of broncho-pneumonia, of dogs that survived the thirty hours, a few died, the average time of death being four days. The latest death was on the ninth day.

Skin lesions. When the vapor of lewisite is applied to the skin, inflammation followed by blisters results. The symptomatology

and pathology of these lesions is very similar to the effects produced by mustard on the skin. After exposure to vapors of lewisite, redness appears in four to six hours, and vesiculation in from sixteen to forty-eight hours depending on the concentration and susceptibility of the individual.

The application of liquid lewisite to the skin also produces burns quite similar to those produced by liquid mustard, though of greater intensity. The action of liquid lewisite differs further from the action of liquid mustard in two important particulars. The latent period is much shorter in the case of liquid lewisite, which begins to burn at once, and causes intense irritation very promptly. Secondly, lewisite differs from mustard and from all other similar compounds that have been used, in that it liberates arsenic after absorption by the skin. Liquid lewisite therefore not only produces a bad burn, but also causes death by arsenical poisoning after the application of comparatively minute amounts—0.02 cc. per square centimeter, per kilo body weight is a lethal dose. Thus a man of average weight (70 kilos) would receive a lethal dose, if 1.4 cc. lewisite was splashed on the skin, if he received no treatment.

When sublethal doses of liquid lewisite are applied to the skin of animals, deep extensive burns are produced, causing spreading necrosis of the skin and subcutaneous tissues. This necrotic tissue is separated by sloughing, and during this process, extensive infection may occur. Completion of sloughing leaves a broad area of hyperemic, dirty infected muscle. Healing then takes place by granulation. The area of necrosis and sloughing in untreated cases is three or four times as large again as the area of application.

When animals receive a lethal application, death occurs in nearly half the cases within twenty-four hours. The rest will die in from two to twelve days. In numerous cases when the application is over the chest wall, hydrothorax is found post-mortem. The lungs and the kidneys are regularly found to be affected. Occasionally there is congestion of the liver, and at other times an intense congestion of the duodenum was found. Less often, subendocardial hemorrhages, suffused valves, and dilated right ventricles are found. The lungs are heavy and voluminous, doughy in consistency, with extensive congestion and edema. Over the surface of the anterior lobes of the lungs are small scattered pink areas of emphysema giving the lungs a mottled appearance. All pulmonary vessels are engorged.

Microscopically, congestion, hemorrhage, edema and atelectasis are found.

The kidneys are of a dark purplish hue with the surface vessels standing out prominently. Capsules strip readily. On section congestion is found in all zones.

On chemical examination arsenic is found in all tissues of the body, but in considerable amounts in the tissues subjacent to the burn, in the liver, kidneys and spleen. Arsenic is regularly excreted in the urine.

Treatment. Since, if lewisite is used, it may very probably be sprayed as a liquid from airplanes, the treatment of these liquid burns which may produce death becomes very important. Briefly, it has been found by experimentation on animals, that the application of hydrolyzing agents immediately after skin burns by liquid lewisite, will preserve life even though they do not prevent the burn. The application of this treatment undoubtedly saves life by preventing the absorption of a lethal quantity of arsenic through the skin. 5 per cent aqueous NaOH solution has been found very efficient. Excision of the burned area and surrounding tissue may be practised up to twelve hours after neutralization of the burn, and the wound will heal by primary union. This indicates that all burns with liquid lewisite should be treated instantaneously, or as quickly as possible with 5 per cent sodium hydroxide solution. After this has been thoroughly applied it should be well washed off with water, as sodium hydroxide is itself irritating to the skin. The patient should be sent to the field hospital as quickly as possible, or at least to a dressing station where simple surgery is possible. There the burned skin should be widely excised and the wound sutured. Excision of the burn may be practised with good results from twelve to twenty-four hours after the burn has been received, if the skin and subcutaneous edematous tissue is removed, and free drainage is provided from the dependent portion of the wound. Even though the wound does not heal by first intention, a great reduction in the duration of the disability will result, as otherwise, ulceration with sloughing and infection are to be expected.

The most suitable treatment for vapor burns has been found to be a ferric hydrate paste prepared as follows: To a solution of ferric chloride slightly under the saturation point, add strong ammonia little by little until a slight odor of ammonia persists. Do not stir,

but place in deep, narrow jars and allow the heavy chocolate colored precipitate to settle to the bottom. Siphon off the supernatant fluid and refill with distilled water repeating this washing until the fluid is free from chlorides as shown by giving no white precipitate on the addition of nitric acid and silver nitrate. The whole washing may require six to eight days. The fine colloidal non granular ferric hydrate so obtained is drained on a filter, and the thick paste is mixed in the proportion of 6 parts ferric hydrate to one of pure glycerine. The resulting smooth paste may be put up in impervious ointment boxes or collapsible tubes. It is very permanent when it is kept in air tight containers.

This paste is applied profusely over the burn, and covered with oiled silk, oiled paper, or other impervious dressing. The dressing may be renewed after about twelve hours. In most cases one overnight application of this treatment prevented blistering, and in many cases it prevented the development of the burn entirely. This paste may also be used to neutralize burns from liquid lewisite immediately after they are received, with operation as soon after as practicable.

Should lewisite be used either by this country or an enemy, ampoules of 5 per cent aqueous solution of sodium hydroxide and collapsible tubes of ferric hydrate paste should form a part of the equipment of all troops in the field. In this way they would be prepared to treat burns at once, after which they should be evacuated promptly for operation at the field hospital. Should it be possible to treat burns in this manner men receiving an otherwise lethal burn should be back on duty in from ten days to three weeks, depending on the intensity of the burn and the promptness with which treatment was applied.

REFERENCES

(1) Lynch, Smith and Marshall: Dichlorethylsulphide, systemic effects and mechanism of action. Jour. Pharmacol. and Exper. Therap., Baltimore, 1918, XII, 265.

(2) Marshall, Lynch and Smith: Variations in susceptibility of skin to dichlorethylsulphide. Jour. Pharmacol. and Exper. Therap., Baltimore, 1918, XII, 291.

(3) Warthin and Weller: The Medical Aspects of Mustard Gas Poisoning. The C. V. Mosby Co., 1919.

(4) Flury and Wieland: Die pharmakologische Wirkung des Dichloräthylsufids. Zeitschr. für die gesamte Experimentelle Medizin, 1921, XIII, 367.

(5) Stewart, Matthew J.: Report on cases of poisoning by "mustard gas," with special reference to the histological changes and to alterations in the leucocyte count. Report No. 17 of the Chemical Warfare Medical Committee, London, December, 1918.
(6) Winternitz: The Pathology of War Gas Poisoning. Yale University Press, 1920.
(7) Wilson and Mackintosh: Mustard gas poisoning. Quarterly Journal of Medicine, 1919–1920, XIII, 201.
(8) Williams: Protective ointments against mustard gas. Journal American Pharmaceutical Assoc., 1919, VIII, 824.
(9) The Official History of the War, vol. II, Medical Services, London, 1923.

CHAPTER IX

The Lachrymators

Lachrymatory gas shell were introduced by the Germans about April, 1915. They were used in considerable numbers until July, 1916, and to some extent throughout the war as harrassing agents. T-stoff shell containing xylyl bromide were first used. The later used B-stoff shell contained at various times xylyl bromide mixed with brom-methyl-ethyl-ketones, bromacetone or cyanogen bromide. The production of lachrymators by the Allies was greatly hampered by the lack of bromine.

These lachrymatory shell produced a certain effect in harrassing the Allied forces especially during the early period of the war when the allied gas masks were relatively inefficient. With the improvement of the gas mask, the only effect of a lachrymator is to cut down efficiency by necessitating the more or less constant wearing of the mask.

At no time did the lachrymators produce more than a few casualties, and these only when an unusually high concentration was produced by the bursting of a shell in a dugout or other confined space, during a bombardment when the men could not escape from the fumes. Such casualties suffered nausea, vomiting and coughing in addition to lachrymation, and in exceedingly rare instances death was caused by pulmonary irritation. Usually these few casualties recovered very promptly when they reached fresh air. In few cases were men incapacitated for more than a day or two.

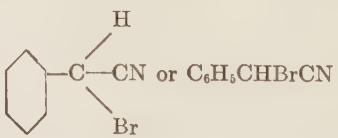
Two fatal cases of poisoning by a lachrymator have been recorded. Shufflebotham (1) reported the case of a workman who was gassed by the explosion of a shell containing ethyl iodoacetate, and died on the same day from severe inflammation of the trachea and lungs. In this case edema was not marked, but in parts of the lungs the alveoli were packed with extravasated red corpuscles. A second fatal case of ethyl iodoacetate poisoning occurred in France in a member of a trench mortar crew owing to the premature explosion of a bomb (2). The post-mortem examination showed pulmonary edema with multiple small hemorrhages in the lungs.

It seems probable that owing to this lack of toxicity, and the protection afforded by the modern mask, lachrymators will be little used in land attacks in future wars. At sea, on the other hand, the tactical value of temporarily incapacitating or even inconveniencing a gun crew might be very great. Moreover, ground that is drenched with a persistent lachrymator would be a very inconvenient spot to occupy, and troops could undoubtedly be harassed in this way during movements to the front or during retreats.

Many chemical compounds possess more or less lachrymatory properties varying in intensity from mild irritation to severe irritation with a flow of tears so copious as to blind the victim temporarily. Some of these compounds, like chlorpicrin, are also toxic. The lachrymatory property seems to be connected with the halogen group and all lachrymators contain one of the halogens. In general, those compounds containing iodine have the strongest lachrymatory effect, bromine is less powerful and chlorine still less effective. Actual effectiveness, however, is only one factor to be considered in the adoption of a chemical warfare compound. It should be cheaply manufactured in large quantities. The comparative rarity and expense of iodine makes the use of this element impractical. Most of the lachrymators used during the war contained bromine and some, like brombenzylcyanide, were extremely effective. But bromine is quite expensive as compared with chlorine, and the development of the lachrymator chloracetophenone, which is as effective as brombenzylcyanide, makes it appear probable that in future no other lachrymatory gas will be manufactured.

BROMBENZYLCYANIDE

Brombenzylcyanide was first discovered in 1881 but was first obtained in the pure state in 1914. It is one of the aromatic side chain halogens having the formula



Pure brombenzylcyanide is a yellowish white crystalline solid

melting at 29°C., and is obtained by recrystallization from alcohol. As commercially prepared the compound contains impurities, and is a heavy, oily, dark-brown liquid. The specific gravity of pure brombenzylcyanide is 1.539 at 0°C. Its vapor pressure has not yet been accurately determined.

Brombenzylcyanide is very stable. Water decomposes it only very slowly. Sodium hydroxide decomposes it very slowly in the cold, but completely after prolonged boiling. An alcoholic solution of sodium hydroxide will decompose it rapidly even in the cold. Some of the stronger oxidizing agents such as chlorates and permanganates will oxidize it after prolonged action.

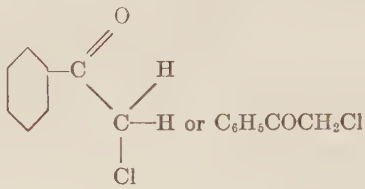
From this stability of composition it may be deduced that it would be very persistent, and this is the fact. Brombenzylcyanide, when spread upon ordinary earth and exposed to the weather would still cause irritation of the eyes and lachrymation after thirty days' exposure. Under favorable circumstances it might last longer. It is certainly one of the most persistent of gases. The stability of brombenzylcyanide is sufficient to permit it to be used in shell when the bursting charge is small. It cannot, however, be used in shell where much high explosive is used, as in armor piercing shell, because it is destroyed in the explosion. For certain tactical purposes this persistence is an objection. It is not as suitable for use against mobs or barricaded criminals as chloracetophenone.

Brombenzylcyanide also has the disadvantage that it attacks the common metals, except lead, quite vigorously. Steel shell are rapidly corroded by it, and in the process the brombenzylcyanide itself decomposes and its lachrymatory properties are lost. For this reason shell intended to contain this substance must be lined with lead, glass or porcelain. This is a serious disadvantage.

Materials splashed with brombenzylcyanide may be cleaned by wiping with a rag soaked in carbon tetrachloride, after removing any water present. Following the use of carbon tetrachloride the last traces may be removed with an alcoholic solution of sodium hydroxide made up in 20 per cent strength. This solution will destroy about two and one-half times its weight of brombenzylcyanide. It is important to destroy all traces of this substance since a very minute quantity can set up an intolerable concentration in the air. Lachrymation is produced by 0.0003 mgm. per liter.

CHLORACETOPHENONE

This compound was first prepared in 1887 but was of no importance until used in chemical warfare. It is a chlorine derivative of an aromatic ketone having the following formula:



Pure chloracetophenone may be obtained by recrystallization from alcohol as white crystals that melt between 50° to 59°C. It boils at 245°C., and can be distilled without decomposition. Its specific gravity is 1.334 at 0°C. It is a solid under normal conditions and has a very low vapor pressure, 0.0028 at 0°C. These properties show that it can be melted and poured into shell or candles. Because of its high boiling point and low vapor pressure, the effect of the compound is lost soon after its initial concentration by condensation to a solid which falls to the ground where it has little or no further effect.

Chloracetophenone is only slightly soluble in water, not to exceed 1 gram per liter, but is soluble in benzene, ether and alcohol, and may be destroyed and removed by a hot aqueous solution of sodium carbonate. Chloracetophenone is an exceedingly stable compound. It is not affected by moisture and may be placed in steel shell without corrosion. It does not decompose on detonation and may even be mixed with T.N.T. It is, therefore, a far more generally useful lachrymator than brombenzylcyanide and is quite as efficient. Lachrymation is produced by a concentration of 0.0003 mgm. per liter. Because of these facts and its cheapness of manufacture, it will probably be the most generally used compound for producing lachrymation. When tear gas is referred to by the general public, it usually refers to chloracetophenone.

Chloracetophenone is used in candles, in which the heat generated vaporizes the chloracetophenone, and also in hand and rifle grenades.

PHYSIOLOGICAL ACTION AND PATHOLOGY

Men exposed to the action of chloracetophenone exhibit the following symptoms. Irritation of the eyes, lachrymation and

CHLORACETOPHENONE SMOKE CANDLES

burning of the more tender portions of the skin. In some cases there is a tendency toward increased salivation and irritation of the throat. Following exposure there may be some discomfort, but this is of relatively short duration, except in the case of exposure to very high concentrations or to low concentrations for prolonged periods. In these latter cases it may require twenty-four hours for the eyes to feel normal again. Men exposed experimentally to high concentrations for short periods of time were unable to open their eyes for several minutes after exposure. In some instances a conjunctivitis was observed which lasted several hours.

Chloracetophenone has comparatively little effect on the skin of either man or animals. When applied in excess to the human skin, there is a burning sensation, slight rubefaction, and sometimes small vesicles appear. This inflammation subsides in about seven days. In vapor form the inflammation produced is very mild and is usually quite unimportant, although in exceptionally sensitive individuals it may prove quite harassing. Chloracetophenone has no effect on the lungs of man in any concentration that will be met with in the field.

Dogs exposed to the toxic action of chloracetophenone exhibit great excitement, irritation of the eyes, nose and throat, lachrymation, salivation, nasal discharge and dyspnea. Vomiting, trembling and twitching of the limbs may occur. Following exposure, there is usually a decided corneal involvement varying from a slight opacity to ulceration and blindness; soreness of the throat and coughing. A few dogs that died showed congestion and edema of the lungs, a true membranous tracheitis, and decided corneal opacity. It is quite impossible that the concentrations used on these animals should be ever encountered in the field. It is stated that horses are quite resistant to chloracetophenone and show no evidence of irritation or lachrymation.

We have no information concerning the mode of action of chloracetophenone or brombenzylcyanide.

TREATMENT

This consists merely in washing the eyes with a solution of boric acid or sodium bicarbonate. As these lachrymators cause no permanent damage to the eyes, and only transient discomfort, even this treatment is generally unnecessary. The mask affords perfect protection against these lachrymators, and with the mask properly

adjusted a man can go through the densest fumes in perfect comfort. However, the effect of lachrymators is practically instantaneous, and some discomfort may be experienced before the mask can be adjusted. The natural instinct when a lachrymator is felt is to turn and run. This is what makes these agents so very effective against mobs and for other police purposes. The first instinct of a soldier with a mask should be to put it on. This is only acquired by training, and when that training has been effective the mask should be on and properly adjusted in seven seconds after the first warning of gas.

REFERENCES

(1) Shufflebotham, Frank: Report on poisoning by ethyl iodoacetate. Report No. 8 of the Chemical Warfare Medical Committee.

(2) Official History of the War, vol. II, p. 465.

CHAPTER X

Irritant Smokes

In the search for toxic compounds that could be used in chemical warfare, arsenical compounds received early consideration. Most of the inorganic compounds of arsenic are solids which cannot be vaporized and are therefore unsuitable for this purpose. Attempts were made to use arsine AsH_3, a very toxic gas. But arsine is inflammable, and is comparatively unstable, and for various reasons was a failure as a chemical warfare agent. However, a considerable number of organic compounds of arsenic were found to be useful.

Diphenylchlorarsine was first used by the Germans in their blue cross shell, July 10, 1917. Later at various times, the blue cross shell contained diphenylcyanarsine, $(C_6H_5)AsCN$, and other arsenical compounds. Ethyl and methyl dichlorarsines and dibromarsines have all been used. The irritant effect of these compounds had been known since 1880. LaCoste describing the action of ethyl-dichlorarsine said, "It has a very powerful irritant action on the mucous membranes of the eyes and nose, causes painful blistering of the skin, and is very dangerous for those working with it, since its vapor causes respiratory embarrassment, faintness, and long lasting paralysis and anaesthesia of the extremities."

But in order to obtain such effects a very considerable dosage is necessary. The arsenical compounds used were all greatly inferior to mustard in vesicant properties and there was no intention to use them for their vesicant or lethal action.

These arsenicals were combined in the shell with high explosive, and it was expected that the shellburst would produce a finely divided cloud of particles which would be small enough to penetrate the mask. Then the violent irritation of the nose and throat and the nausea which is produced by these agents would necessitate the removal of the mask, when the victim would succumb to other more lethal gases. Thus these arsenical compounds were first known under the name of sternutators, or sneeze gas. But dispersed in this way the particles were, as a rule, too large to penetrate the mask, and the blue cross shell was, comparatively speaking, a failure.

The Germans overestimated the value of their blue cross shells and produced them in enormous numbers. The high estimate they placed on the blue cross shell can be seen from the following figures:

Captured German divisional shell dump, Moreuil sector, July, 1918

	per cent
H.E. shell	50
Blue cross shell	19
Green cross (lung irritants)	24
Yellow cross (mustard)	7

Captured German shell dumps opposite British 3rd Army, September 1918

	per cent
H.E. and shrapnel	60
Blue cross	30
Other gas shells	10

The following instructions issued from the German general headquarters under Ludendorff's signature on July 9, 1918, express the German view of blue cross shell,

> The substance in blue cross shell acts with extraordinary rapidity, in fact almost instantaneously, but it generally puts troops out of action for only a short time. In sufficient concentrations it penetrates the French mask effectively and the English mask to a lesser degree, in which case it forces the enemy to tear off their masks. For this reason a mixture of blue and green cross is recommended. In the case of an insufficient concentration, blue cross at least forces the enemy to wear their masks, thereby interfering with their fighting efficiency. The effect of the gas passes away and rapidly disappears after the gas or detonation cloud. In consequence it can be employed when our infantry are relatively close to it.

It has since been found that the compounds of this class may be dispersed by heat as smokes. The compound is liberated from the container as a vapor, which condenses in the air, thus forming very minute particles which are more difficult to filter out in the mask and and are more toxic. A much higher concentration can be maintained by very fine particles, and the finer the particles the more quickly are they absorbed from the lungs. Also, the finer the particles, the more lasting is the smoke.

Although many organic arsenical compounds lend themselves to this use, there are two compounds that have been found superior and are now generally used. These are diphenylchlorarsine, for convenience called D.A. and diphenylaminechlorarsine, known as D.M.

D.A. and D.M. are almost identical in tactical effect but differ in minor details.

SMOKE CLOUD PRODUCED BY CANDLES

With a very slow wind and consequent slow spreading of the cloud, D.M. from only 500 candles on a front of 100 yards was effective to the point of producing vomiting among unprotected subjects three miles from the origin and detectable in irritating approximately six miles from the source.

DIPHENYLCHLORARSINE, OR D.A.

D.A. was discovered by LaCoste and Michaelis in 1878. The work of Michaelis which was conducted originally with a view to the medicinal effect of arsenical compounds, later led to the discovery of atoxyl, salvarsan and similar compounds. Diphenylchlorarsine has the following formula:

$(C_6H_5)_2AsCl$ or

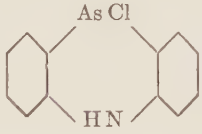
It may be regarded as a derivative of arsine, AsH_3 by replacing two hydrogen atoms by two phenyl groups, and the third hydrogen atom by chlorine.

Pure D.A. is a colorless, crystalline solid that melts at 44° to 45°C. The plant product is a thick semi-solid mass. D.A. cannot be distilled in air without decomposition. Its boiling point in an atmosphere of carbon dioxide is 383°C. The vapor pressure of D.A. is 0.0001 at 0°C., and 0.0004 at 25°C. This vapor pressure is negligible, and the compound cannot be vaporized at ordinary temperatures. D.A. is not decomposed by detonation. D.A. is slightly soluble in water. It is freely soluble in phosgene and carbon tetrachloride, mineral oil and nearly all organic solvents. It may therefore be readily mixed with other chemical warfare agents, such as phosgene or chlorpicrin. Chlorine, however, unites with D.A. to form a non-toxic product. D.A. is hydrolyzed by water forming diphenylarsenic acid, but the process is very slow in moist air. D.A. is unchanged by exposure to air at ordinary temperatures.

DIPHENYLAMINECHLORARSINE, OR D.M.

D.M. was discovered in 1918 independently in this country and in England. The American discoverer was Dr. Adams, and the compound is sometimes called "adamsite." The formula of D.M. is:

$(C_6H_4)_2HN:AsCl$ or



Pure D.M. is a canary yellow crystalline solid. The crude product is a dark green or brown crystalline solid. The melting point of D.M. is 195°C., and the boiling point is 410°C. It decomposes on boiling at atmospheric pressure. D.M. is more insoluble than D.A. It is only moderately soluble in common organic solvents. It is nearly insoluble in phosgene, and it probably cannot be successfully mixed with other liquid chemical warfare agents. D.M. acts on metals, rusting iron and steel and tarnishing bronze and brass. D.A. does not act in this way. On the other hand D.M. is much easier to manufacture than D.A., and its toxic effects are of longer duration.

SYMPTOMS PRODUCED BY TOXIC SMOKES

The effects of the various arsenical toxic smokes are very similar although there are minor differences in their action. Thus methyl dichlorarsine is more lethal for dogs than diphenylchlorarsine, but less irritant to man, therefore D.A. and D.M. have been most used, not because they are the most lethal agents of this type, but because they are among the most highly irritant.

When animals are exposed to lethal concentrations of these arsenical compounds, the following symptoms are commonly noted. The animals show some excitement, followed by slight lachrymation, some drooling of saliva, and towards the end of the exposure respiration may be increased in frequency and depth. There is practically complete absence of the symptoms of tremendous irritation which are present in men exposed to much smaller concentrations, and no evidence of marked pain, retching and vomiting or loss of the sense of balance. Following the exposure there may be vomiting, increased nasal and buccal secretion, and swelling of the eyelids with conjunctivitis. Sensory and motor disturbances are practically never found, in which respect again animals react to these compounds differently from man.

Depending upon the concentration and length of exposure, the animals may die as early as twelve hours or as late as twenty days after gassing.

When dogs die as the result of exposure to low concentrations, the respiratory system is found to be normal throughout. The pericardium is filled with an amber colored fluid which usually contains some stringy exudate. When dogs die as the result of exposure to higher concentrations an intense involvement of the entire respira-

AIRPLANE PRODUCING SMOKE

tory system is noted. The larynx and trachea are both affected containing more or less yellowish purulent exudate. The trachea may be almost filled with this exudate or with blood tinged fluid. On cutting the trachea this exudate adheres to the walls. The involvement of the mucous membrane extends down into the bronchi, where exudate and fluid may occlude the lumen.

The lungs are voluminous and heavier and firmer than normal with areas of edema and congestion everywhere. Scattered areas of focal pneumonia, with or without abscess formation may be found. Microscopically there are found edema, marginal areas of atelectasis, congestion of the parenchyma, congestion and swelling of the alveolar walls, engorgement of the capillaries, and distention of the perivascular spaces. Fibrin and leucocytes line the bronchial epithelium which is generally intact. The abdominal organs are generally normal, though congestion of the liver and spleen and congestion and ulceration of the gastric mucosa may be seen in exceptional cases. None of the animals showed any demonstrable changes in the nervous system. It is to be noted that although there are many scattered areas of edema in the lungs of such animals, the injury to the alveolar area is much less marked than is the case following gassing by the pulmonary irritants, the principal damage being to the bronchi.

Analysis of the organs of such animals shows that arsenic is absorbed and distributed throughout the body. The following examples illustrate this distribution.

Dog E.A.M.R.D. 1326, Field test, exposed to D.M. smoke, concentration 0.184 mgm/liter for four minutes. Killed with magnesium sulphate on eleventh day. Organs extracted by Kjeldahl method and then the arsenic estimated as As_2O_3 by the Gutzeit method.

	Milligram to gram of organ
Brain	0.001
Heart	0.002
Kidney	0.008
Liver	0.007
Pancreas	0.005
Spinal cord	0.001
Spleen	0.0015

Dogs exposed in gassing chamber, thirty minutes to various concentrations of D.A.

Dog E.A.M.R.D. 1304, concentration 0.262 mgm./liter, death in thirteen days.

	Milligram to gram of organ
Liver	0.013
Kidney	0.004
Spleen	0.002

Dog E.A.M.R.D. 1321, concentration 0.49 mgm./liter, death in twelve hours.

	Milligram to gram of organ
Sample 1 brain	0.015
Sample 2 brain	0.014
Spinal cord	0.005

Dog E.A.M.R.D. 1325, concentration 0.49 mgm./liter, death in twelve hours.

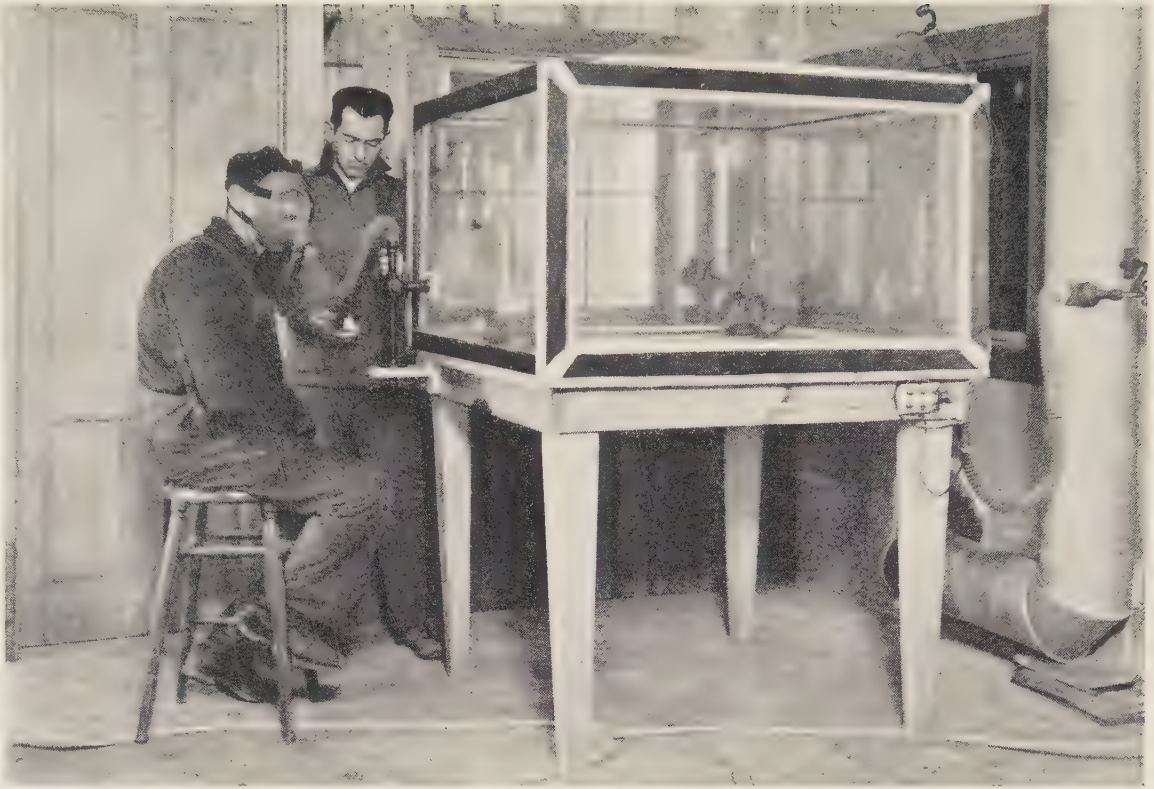
	Milligram to gram of organ
Brain	0.0016
Heart	0.003
Kidney	0.020
Liver	0.014
Pancreas	0.011
Spinal cord	0.001
Spleen	0.002

SYMPTOMS PRODUCED IN MAN BY TOXIC SMOKES

When men are exposed to the toxic action of minute amounts of D.A. under laboratory conditions the following effects are noted in order of appearance: Irritation of the nose and throat; irritation of the eyes with lachrymation; irritation of the chest with a growing feeling of suffocation; excessive nasal secretion, ropy saliva and nausea. If exposure is prolonged, aching pains in the stomach and numbness of the limbs may appear. This numbness may later change to extremely sharp pains.

Subsequent to exposure the following symptoms appear; a slight dizziness immediately after exposure; profuse salivation; intense burning in the nose and throat; occasional nausea, headache and toothache. The burning in the nose and throat persists after all other effects have disappeared. Usually these delayed effects are mild. The period of incapacitation after even a rather high concentration of diphenylchlorarsine is very short, rarely lasting longer than thirty minutes, and is caused by nausea, dizziness and headache.

A comparison of the effects of D.M. and D.A. is as follows: During the time of exposure the effects of D.A. are more apparent than those of D.M. With the higher concentrations, the effects of D.A. were more immediate and just as severe, but not nearly so persistent as



APPARATUS USED IN TESTING IRRITANT SMOKES ON MAN

those of D.M. With lower concentrations the effects of D.A. were neither as severe or as lasting as those of D.M.

With neither of these smokes does the immediate effect give adequate warning of the more severe after effects. Practically the largest period of incapacitation due to D.A. was one-half hour, whereas with the higher concentrations of D.M., cases of two to three hours incapacitation were frequent. D.M. would appear to be more efficient since from its lighter immediate effects men would remain longer before seeking relief, and would thus be incapacitated for several hours following the exposure.

No figures are available to show the lethal concentration of the various arsenicals for man. It is highly improbable that death will be caused by the use of these materials under field conditions. The American mask affords practically complete protection against these smokes, and should masks not be available or should they be penetrated, the irritating properties of these smokes are so intense that troops will be compelled to break and run long before lethal concentrations are attained; 0.0015 to 0.0025 mgm. of D.A. per liter are sufficient to produce irritation of the nose and throat. 0.05 mgm. per liter will prove intolerable in half a minute. Yet it requires between 0.1 to 0.2 mgm. per liter to cause the death of dogs after an exposure of thirty minutes. D.M. is not lethal even for animals.

However, considerable temporary disability may be produced under field conditions by the use of these smokes. The following description gives the results of the use of these agents during the war.

When a man was seriously affected by the fumes arising from a blue cross or green cross 3 shell the effects began to show themselves within a few moments of exposure and developed in rapid sequence: Intense pain of an aching or burning character in the nose, mouth and throat, pain in the gums and jaws, tingling and smarting of the face, especially the cheeks and lips, aching pain in the eyes and frontal headache, watering of the eyes and painful conjunctivitis, copious watery discharge from the nose, tightness and burning sensation in the chest, salivation, pain in the stomach, nausea, retching and sometimes vomiting which might be followed by tenesmus, though diarrhea was practically never met with. The pain in the nose and throat was not infrequently described as passing upwards into the eyes and head, a fact suggestive of the involvement of the frontal and accessory sinuses. It is by no means easy to describe this pain. If slight it resembled in some degree the pain which is sometimes experienced when fresh water gets into the nasal sinuses whilst bathing, but in the more severe cases it was of distressing intensity and sometimes agonizing. The pain in the chest was always described as

of a burning character in the early stages. The degree to which the different symptoms developed differed even in the severe cases. Thus in some cases little complaint was made of distressing pain in the nose or head, and the throat felt unpleasantly dry rather than painful, though an intense burning sensation was felt in the chest. Possibly these differences were dependent on differences in the mode of breathing, for the serious involvement of the nasal passages would be less likely to occur in the case of mouth-breathers. The pain in the eyes was somewhat different from the smarting caused by the simple lachrymators, the impression being gained that small gritty particles had been driven into the eyes. At an early stage when the distress was at its height the skin of the cheeks, especially close to the eyes and nose, might feel tense to the patient and have a slightly swollen look. Sneezing appeared to characterize the mild rather than the severe cases.

A very early condition sometimes complained of was that of giddiness, and a certain number of cases quickly lost consciousness, a comatose condition persisting for several hours. Others in whom consciousness was never lost passed into a lethargic condition for a period of twelve to twenty-four hours. A remarkable feature in these severe cases was the intense mental distress which accompanied the symptoms already described. Even the slighter cases felt and looked miserable and wretched until the irritation had passed off, and the picture of utter dejection and hopeless misery furnished by the severe cases had no counterpart in any other type of gas poisoning. Occasionally the psychical depression resulted in the temporary loss of mental control, and men were in a few instances reported as having acted as if they were driven mad by their pain and misery. In at least one instance a man in this condition had to be forcibly restrained from shooting himself, and in another case a man tried to get under the floor of a hut under the delusion that he was being pursued.

In addition to these changes in the mental condition, alterations in motor power were not infrequent. They might supervene within an hour or be more delayed in appearance. Though the commonest complaint was of formication mainly referred to the finger-tips—and this has also been observed in laboratory workers who would not be likely to exhibit functional phenomena—yet temporary paralysis involving one or more limbs was seen in a fair number of cases. In one instance complete paralysis of the left arm developed within three-quarters of an hour after exposure, so that by the time the man reached the advanced dressing station his arm was hanging like a flail and useless. By the time, however, that he had arrived at the casualty clearing station, an interval of about four hours, this paralysis had almost entirely disappeared, and within twenty-four hours there was no sign left of it. Such instances as these were proportionally infrequent, though they were a strong argument in favor of a central toxic action of the gas which affected the cortical or spinal centers leading to a temporary abolition of function. Again, other instances occurred in which a more generalized motor weakness appeared, and one well-authenticated example of the combination of this motor weakness with mental depression may be quoted. A party of men had been affected by blue cross gas and in addition to the irritative phenomena already described they developed a considerable weakness of the legs so that progression was

difficult and uncertain. After taking a few steps the men would fall down. They would very shortly rise and struggle forward again. There seemed very little reasoning power in their progression, and they presented a picture of a loss of mental control which would not permit them to lie still when they fell but forced them to struggle on despite the motor weakness from which they were suffering (1).

The experience of the Germans seems to have been similar, as is shown by the following quotation from Flury (2):

In addition to the phenomena of sensory irritation, inhalation of diphenylcholarsine may lead to serious disturbances of the nervous system from absorption of the poison. These show themselves as motor disturbances, uncertain gait, swaying when standing, and sometimes complete inability to walk. As a rule they are accompanied by severe pain in the joints and limbs. Inhalation of very high concentrations is also often followed by giddiness, attacks of faintness, and loss of consciousness, which may last for many hours. When considerable quantities or diphenyl-chlorarsine or related organic arsenical compounds are taken through the skin nervous disturbances of various types may arise, and these are to be ascribed not to a local action of the poison but to its general absorption. Hyperesthesia, anesthesia and paresthesia of definite areas of the skin, especially of the lower extremities, could frequently be observed. Twitching of the muscles and convulsions may occur in very severe cases of poisoning by similar substances.

In a small percentage of cases, these immediate symptoms, which may be ascribed to toxemia affecting the nervous system, are followed after an interval of several days by marked sensory disturbances. Anesthesia, varying from mere numbness of the finger tips to a complete loss of sensation over a considerable part of one or more limbs, was occasionally observed. Commonly this anaesthesia was bilateral and was of the glove or stocking distribution. The reflexes in these cases were normal, pressure on the nerve tracts was not painful, nor were there any other signs of peripheral neuritis. It was believed that in these cases, the sensory changes were functional in character. It could hardly be supposed that arsenical peripheral neuritis would affect the sensory nerves alone, and the stocking distribution of anesthesia is rather indicative of functional disorder. Recovery from this condition was rapid and uniform.

But such cases are quite exceptional. The vast majority of patients gassed by blue cross shell during the war were incapacitated only a few hours, and were able to return to duty in from twenty-four to forty-eight hours, so that the British Army made it a rule that such cases should be retained in the zone of the advance.

The number of casualties that may be expected from the use of these irritant smokes will depend chiefly upon the effectiveness of

gas discipline, but may be considerable. Thus in the British fifth crops gas center, from May 25 to October 9 1918, out of a total of 3510 cases admitted, 9.6 per cent were blue cross casualties, while in the same hospital between May 25 and June 30, the percentage increased to 17.3 per cent following a decided increase in blue cross shelling. It is evident that although the Germans overestimated the value of these compounds, especially as employed in explosive shell, they were good casualty producers, and probably lowered the morale out of all proportion to their casualty producing powers. However it must be remembered that although methods of distribution of toxic smokes have been greatly improved, the mask also has been improved, and will now keep out much finer particles. Protection resolves itself into a question of gas mask drill and gas discipline.

The diagnosis of these cases is usually easy. The symptoms of violent irritation of the nose and throat appearing practically immediately after exposure are characteristic. Lachrymators have little effect on mucous membranes of the nose and throat, and after exposure to practically all other gases, there is a definite latent period before the development of symptoms. But the enemy may very probably use other gases together with arsenical irritants, and the possibility that other symptoms may develop later must always be borne in mind. The use of pulmonary irritants is most to be feared at such times.

Treatment. Rest, fresh air, and removal of contaminated clothing. Very minute amounts of these substances in clothes, or around shelters will cause persistence of symptoms. Wash the nose and throat with saturated solution of boric acid, or with physiologic saline solution. Relief for the burning of the nose and throat is afforded by inhaling the following mixture:

Alcohol	40 per cent
Chloroform	40 per cent
Ether	20 per cent
Ammonia	5 to 10 drops

The ammonia may also cause some relief from the nausea and depression. Glycerine and menthol troches help to allay the burning of the throat. Phenacetin, aspirin and similar preparations may be used to relieve headache and other pains, and a single dose of morphine will probably relieve the worst of the symptoms. Morphine is not counter-indicated as in poisoning with pulmonary irritants.

No treatment other than symptomatic is required, since nearly all victims will make a complete recovery in the course of a day or two. Even the few cases suffering muscular weakness and anesthesias require only rest with graduated exercises and sufficient occupation for the mind, bearing in mind the fact that the disability is functional rather than organic.

Such stable arsenical compounds as D.A. and D.M. may readily contaminate water and cause poisoning if such water is used for drinking or cooking. This occurred on several occasions during the war as shown by the following example:

Two officers and thirty-one other ranks of No. 2 Coy. obtained water from a shell-hole for washing and shaving. After doing so they complained of great burning in the eyes and gums, which subsequently developed into swelling of the mouth and conjunctivitis. Those who only washed in the water were affected by a splitting headache, intense stinging of the face, swelling up of the cheeks, marked lachrymation, and soreness of the throat. Men who washed their teeth in the water complained of acute burning in the gums. Two or three who drank the water suffered from vomiting. Water obtained from the shell-hole in question was found to be heavily contaminated with arsenic in organic combination, equivalent in amount to 3.75 mgm. of As_2O_3 per liter. No. 1 Coy., who were immediately on the flank of No. 2 Coy., had no casualties, but they drew their water from the water-cart.

It is necessary to issue orders that no water from shell-holes or other sources open to contamination be used. To identify arsenic under such circumstances, the organic combination must be broken down. The following method may be used:

Evaporate 1 liter of the suspected water to dryness on the water bath, transfer the solid residue to a long-necked hard glass flask (Kjeldahl flask) add 1 cc. of concentrated nitric acid (or 2cc. if there is much chalk present) and 25 cc. of concentrated sulphuric acid. Heat over asbestos gauze for at least six hours at a temperature just below the boiling point of the sulphuric acid. Keep the flask inclined to prevent spurting. Allow to cool and dilute to 250 cc. with distilled water. Pass sulphur dioxide through the solution for twenty minutes to reduce the arsenic acid to arsenious acid. Boil off the sulphur dioxide and test the solution by Marsh's method, or better by the Gutzeit test.

REFERENCES

(1) Official History of the War, vol. II, p. 475–480.

(2) Flury: Lokal reizende Arsenverbindungen. Chapter IX, p. 523. Zeitschrift für die gesamte Experimentelle Medizin, Band XIII, Berlin, 1921.

(3) Lefebure: The Riddle of the Rhine 1921. W. Collins & Sons, Glasgow.

CHAPTER XI

Miscellaneous Gases

HYDROCYANIC ACID

Hydrocyanic acid is a paralysant gas whose effect is directly due to its toxic action on the nervous system. It was used by the French in shells under the name Vincennite, and was also used experimentally by the British. The Germans did not use it. It was impossible to obtain a lethal concentration under field conditions, when firing it in small shell, and its use was never extended. It deserves study because it has been used and may be used again under different circumstances.

Physiological action. The majority of the cyanides are toxic in proportion to the amount of CN they contain. As potassium cyanide and sodium cyanide are more easily handled than hydrocyanic acid gas they are the compounds usually fed or injected intravenously in laboratory experiments. As a result of such experiments Sollmann (1) summarizes the action of HCN as follows:

> Hydrocyanic acid is probably the most rapidly acting poison. It produces death under asphyxial symptoms, by hindering the oxidative processes of the tissues. It also interferes with most other fermentations and is therefore a general protoplasmic poison. Compounds containing the CN radicals are toxic only if they can liberate HCN. Ferro-, ferri, and sulphocyanides are stable and therefore do not show these effects. When given in doses small enough to permit of watching its action, this is seen to consist in a fleeting stimulation of certain parts of the central nervous system, followed by depression and paralysis. The action begins in the medulla. The vomiting, respiratory, vagus, pupil-dilator, and vasomotor centers are all stimulated. Then comes unconsciousness, and after this convulsions. In man these are probably mainly, medullary in origin. Then follows paralysis of the whole central nervous system. Involuntary evacuations of feces, urine, and semen are frequent.

Effects on special tissues and functions. 1. The blood. The cyanide does not combine with the hemoglobin. Cyan-hemoglobin and cyan-oxyhemoglobin are only formed after several hours exposure and with difficulty. The small trace formed can only be due to laking of the corpuscles: intact red cells will not take up CN. We

have been unable to demonstrate cyanogen in the blood of our animals, even on using a large amount and distilling it, and it is reasonable to presume that it is not present in any detectable quantity. The bright color of the blood in poisoning is due to the fact that the venous blood contains an excess of oxygen over the normal. In other words the tissues have been unable to remove the oxygen from the blood. The oxygen in the blood is more readily dissociated than in the normal. This is due to a diminution of the alkali reserve which accompanies the anoxemic formation of fixed acid and in cyanide poisoning the blood has been shown to contain a slight excess of fixed acid. This acid, lactic acid, has been found both in the blood and urine of animals poisoned by HCN.

2. The blood-vessels. There is an early rise of the blood pressure due partly to central stimulation and partly to an increased outpouring of secretion from the suprarenal glands. This initial rise is soon followed by a marked fall due to vaso-motor paralysis and also to direct action of CN on the vessel walls. The weakening of the heart beat due to oxygen lack and direct action of the poison also contributes largely to the fall in blood pressure.

3. Heart. In low concentrations cyanides do not affect the isolated heart greatly. With stronger solutions a weakening of the force of contractions is seen, also an alternation of beats and finally a heart-block similar to that seen in asphyxia. In injected animals a similar effect is seen—first a slowing of the beats due in the early stages to central vagal stimulation and in the later stages to heart-block.

4. Respiration. Small doses whether by inhalation or injection cause quicker and deeper respirations. Later the respirations become slower and shallower, with prolonged pauses (Cheyne-Stokes type). The above changes are caused by a primary stimulation followed by a later paralysis of the respiratory center. With large doses the immediate cause of death is this respiratory paralysis as the heart continues to beat after respirations have ceased. The gas is not irritant to the lungs and the above effects on the respirations occur in vagotomized dogs.

5. Muscle tissue. Cyanides have a marked effect on plain muscle, causing the rhythmic motions of isolated intestinal loops to cease. In low concentrations there is no marked effect on striped muscles other than a slowing of the rate of recovery from fatigue. Stronger concentrations cause immediate death of the tissue but this effect is never seen in poisoning cases.

6. Nervous system. The central nervous system is first stimulated and then depressed. The spinal cord centers are affected less and recover first. The main action is on the medulla oblongata and lower brain. The inhibitory influence on the respiratory center is marked from the first, but later the whole central system is paralyzed. The early convulsions are due to a direct stimulating effect on the higher centers, the later ones are probably asphyxial in origin.

7. Action on ferments. In general, cyanides have a marked influence on ferments and the lower forms of life, inhibiting such varied phenomena as the action of blood on hydrogen peroxide, the activity of platinum sponge black, the growth of yeasts and plants and the germination of seeds.

8. Excretion. A certain amount is excreted by the lungs. The remainder is excreted as sulphocyanides after being bound in the body to sulphur containing molecules.

Theories of action. The action of hydrocyanic acid on the tissues as noted above is practically the same as that of asphyxia. The marked difference between the two conditions is the presence of an excess of oxygen in the blood in cyanide poisoning. The tissues are suffering from lack of oxygen but it is an internal asphyxia, undergone in the presence of an abundance of oxygen which they cannot utilize. This failure to utilize the oxygen may be due

1. To the HCN inactivating the oxygen.
2. To an effect on the intracellular enzymes generally.
3. Most probable of all, to a chemical action on some part of the oxidase system, probably the autoxidisable substances. These contain SH and cyanogen is excreted as sulphocyanates.

While the general tendency is to ascribe the whole effect of the cyanides to their action on the oxidation processes, the immediate inhibition of the respiratory center by doses that do not greatly affect the oxidative processes in other organs suggests that hydrocyanic acid may have a specific action on this center.

Lethal dose. This is usually given as 0.05 gram of absolute HCN for man. The lethal dose by respiration is usually believed to be less than the above and our figures confirm this view.

Symptoms. In an atmosphere containing a lethal concentration an odor of bitter almonds is noticed. This is followed rapidly by a sensation of constriction of the throat, giddiness, confusion, and indistinct sight. The head feels as though the temples were gripped in a vice, and there may be pain in the back of the neck, pain in the

chest, with palpitation and labored respiration. Unconsciousness occurs and the man drops. From this moment if the subject remains in the atmosphere of hydrocyanic acid more than two or three minutes death almost always ensues, after a brief period of convulsions followed by failure of the respiration. The heart beats some time after respirations have ceased, becoming weaker and weaker until it stops completely. If the man can be removed from the gas at the moment he drops, and can breathe uncontaminated air he will usually recover. In exceptional cases there is a more or less transitory paralysis of the lower limbs.

In experimental animals we have found that there was an upper limit of low concentrations where all animals promptly recovered, and a lower limit of high concentrations in which all the animals promptly died. However between these two limits was an intermediate zone of concentrations in which the animal might die at once, might recover completely, or might live for several days only to die finally. These latter animals lay prostrate in the cages, breathing slowly but showing no other signs of life. They were totally unable to eat or drink even when water was held in front of them. In certain instances a continuous swimming or clawing motion was made with the forelegs. This was probably caused by irritation of the nerve centers. These animals undoubtedly suffered from a permanent injury to the nerve centers of the brain and cord. This permanent paralysis has never been described in human cases. It is generally stated that if death does not occur within an hour that recovery will be prompt and complete. Experimental animals that have recovered have often been able to walk about at the end of an hour. But it is safe to say that men who have lost consciousness after inhaling hydrocyanic acid, will not be capable of effective defense for several hours. In some cases not for several days.

Post mortem findings. At necropsy one finds scarcely anything. The organs are tinted pink or bright red if the post morten is held promptly. This is the most significant finding since it indicates a high degree of oxygenation of the blood. The lungs are pink and diminished in volume. The heart is found in systole. The blood is bright red, slightly coagulated. There are practically no other organic changes.

Treatment. On account of the rapidity of action, there can be little treatment. The individual must be removed immediately

from the gas to an uncontaminated atmosphere if possible. Artificial respiration may be used if the respiration has ceased. Application of cold water to the nape of the neck, and injections of ether or caffeine are recommended. If the respiration is resumed further treatment is unnecessary.

CARBON MONOXIDE

Carbon monoxide has never been used as a war gas and such use in the future is highly improbable. However during the past war many men were overcome by carbon monoxide. Practically all explosives produce enormous quantities of carbon monoxide. Ordinary gunpowder produces more than any other explosive, namely, from 40 to 60 per cent depending upon the rapidity and completeness of detonation; but ammonal, trinitro toluene and other high explosives all produce large quantities. It is stated that 1 kgm. of such explosive may generate 800 liters of CO.

Carbon monoxide intoxication therefore occurs within closed spaces under the following conditions:

1. When guns are fired from loop holes of a closed shelter. In the absence of adequate ventilation, the atmosphere rapidly becomes toxic.

2. When a mine or camouflet explodes. The surrounding galleries and communication trenches may become untenable, and the process of digging out caved in earth works is dangerous because of the pockets of gas that are liberated.

3. When a projectile bursts in a dugout, or even in the ground near it. The ground and wall of the explosion crater are impregnated with the evolved gases, and these may filter out slowly into the dugout.

4. Dugouts and improperly ventilated shelters may accumulate the carbon monoxide given off by fires, especially from charcoal braziers.

Physiological action of carbon monoxide. Carbon monoxide forms a firm union with hemoglobin, thereby preventing it from carrying oxygen and producing asphyxia. The fatal results of simple anoxemia may be readily observed in carbon monoxide poisoning, since CO has no direct action of any kind.

Carbon monoxide hemoglobin is a very firm compound. It is distinguished spectroscopically, is not reduced by ammonium sulphide, and has a cherry red color very different from oxyhemoglobin. This color is a good test for its presence.

The affinity of CO for hemoglobin is variously given as from 210 to 250 times as great as that for oxygen, and as a result very small proportions of this gas will unite continuously with the hemoglobin and produce asphyxia. The amount of CO absorbed is proportional to the amount in the air. Toxicity begins with about 0.05 per cent or 5 parts per 10,000, the atmosphere is dangerous with 0.16 per cent and 0.2 to 0.4 per cent is generally fatal. Dizziness and labored breathing occur when about 20 per cent of the hemoglobin is saturated, unconsciousness with 50 per cent and death when 60 to 80 per cent saturation is reached.

Symptoms. The symptoms of acute poisoning may be divided into three states (1) until loss of consciousness; (2) from loss of consciousness to onset of apnea; (3) coma with arrested respiration.

1. The onset depends upon the rapidity with which CO is absorbed. When poisoning takes place rather slowly the symptoms begin with dizziness, headache, weakness particularly of the legs, nausea and vomiting. This is followed by drowsiness, stupor and unconsciousness. With massive doses the loss of consciousness is very rapid. The first symptom may be violent headache with a sense of constriction at the temples, ringing of the ears and a sensation of fog before the eyes. The subject rises and endeavors to escape but is seized by vertigo, staggers and falls.

2. After unconsciousness, the respiration becomes accelerated and sterterous, the skin cyanotic. Reflexes and sphincter control are lost and there is then a period of convulsion. The pulse and respiration gradually weaken, ending in apnea. This stage may be considerably prolonged because as the victim ceases to breathe, little additional carbon monoxide is absorbed.

3. Coma sets in and death advances steadily but slowly because respiration is reduced to a minimum. Finally the heart is arrested.

Mild intoxication is generally found in men unable to leave a contaminated dugout, or among men who have been able to leave a dangerous atmosphere promptly. The patient presents a stupefied aspect and his red face and staggering gait suggest drunkenness. The patient complains of splitting headache, foggy vision, ringing in the ears, weakness, vertigo and nausea.

In some cases the first and main symptom is an utter weakness and powerlessness of the legs. In such cases men may be aware of the danger and unable to escape. Should the man fall and endeavor to work his way out with the hands, this only increases the rapidity of

action of the poison should he fail to escape, for the effort increases the respiration. It is well known that in numerous instances men have been overcome through running their engines in a closed garage, and an automobile engine may produce from one to two cubic feet of carbon monoxide per minute, and a car warming up in a closed garage of 1500 to 2000 cubic feet with the doors closed makes an atmosphere dangerous to life in five minutes or less. The dangers from leakage of illuminating gas are well known. At Edgewood Arsenal the illuminating gas contains small quantities of butyl mercaptan, one of the substances that makes the skunk so malodorous. Leaks in the gas can therefore be detected promptly although the gas itself is odorless. Too much can hardly be said concerning the insidious nature of this gas, which is tasteless, odorless and non-irritating.

Pathology. When exposure to the gas has resulted in a high degree of saturation of the blood, the skin especially of the face, chest, shoulders and thighs may be colored bright red. On section, the blood, and muscles and the organs generally, particularly the lungs and liver are a bright cherry red. There are practically no other post-mortem changes except in the brain. In cases where the poisoning has been prolonged the brain is seriously affected. There is great engorgement and congestion of the vessels of the meninges and brain. The intense congestion generally produces a variable degree of edema of the brain. In the cortex, in addition to this congestion many capillaries may be thrombosed, and minute punctiform hemorrhages are found. These hemorrhages are quite characteristic of severe anoxemia and are also found in cases dying of pulmonary edema following phosgene poisoning. These same vascular changes and hemorrhages are found through other parts of the brain, especially in the centrum ovale, internal and external capsules and optic thalamus. There may be degenerative changes in the cells surrounding these hemorrhagic areas and in cases of late death areas of necrosis may be found. These changes sufficiently indicate the damage produced in the brain by prolonged anoxemia, and explain the fact that recovery from serious carbon monoxide intoxication is slow and that the functions of the brain are frequently permanently impaired.

Treatment. In the presence of a large excess of oxygen the CO hemoglobin is decomposed. The treatment is simple. Remove patient from the gas. If respirations have ceased, begin artificial

respiration at once by the Schaeffer or prone pressure system. As soon as respiration is established administer pure oxygen. Haggard and Henderson (2) recommend the administration of oxygen containing 8 per cent carbon dioxide, which stimulates the respiratory center. By inhalation of this mixture the breathing is quickly restored to normal, or more than normal amount. Wrap the patient warmly with hot water bottles. One of the characteristic effects of CO poisoning is lowering of the body temperature. The patient often complains of cold and the extremities and surface of the body are cold to the touch. Even with respiration of ordinary air the CO hemoglobin is decomposed below the danger limit in an hour. It is therefore unnecessary to administer oxygen longer than one hour. CO is practically all eliminated in two to three hours.

After effects. Complete recovery is slow. As Haldane said, "Anoxemia not only stops the machine, but wrecks the machinery." Recovery from the acute effects generally occurs in forty-eight to seventy-two hours but convalescence is not complete. The brain especially is affected by the anoxemia. Accordingly some of these patients remain drowsy. Later some of these patients are troubled with obstinate insomnia. Persistent headaches, mental changes, loss of memory, insanity, or various forms of paralysis are common sequels.

Prophylaxis. The gas mask does not afford protection against CO as the canister is only designed to protect against war gases. Therefore in any case where an atmosphere vitiated by carbon monoxide must be entered, oxygen helmets such as used in mine rescue work must be used.

When men are working in mines, or other locations where they may encounter carbon monoxide, mice or better canaries should be carried. The metabolism of these small creatures is many times more rapid than that of man and they therefore succumb to the effects of carbon monoxide long before men are affected. If the bird or mouse becomes unconscious, the working party should leave at once if they are not provided with oxygen breathing apparatus, for if they remain they will in time be overcome likewise.

ARSINE (ARSENIURETTED HYDROGEN) AsH_3

General properties. A colorless gas having a marked and disagreeable garlic like odor. Slightly soluble in water. Molecular weight 78; weight of a liter of gas is 3.48 grams, and its density

relative to air is 2.69. It may be liquefied, and the liquid is stable and without effect on lead or steel. It is quite toxic so that a few bubbles of gas may be fatal to man.

Arsine was not used as a war gas though it might be so used under certain circumstances. It is produced in lead burning and by the action of impure sulphuric acid on metals. All commercial sulphuric acid contains arsenic. The best grades contain about 0.0000025 per cent arsenic, and cheaper grades may contain considerable percentages. When sulphuric acid containing a high percentage of arsenic is used in storage batteries in enclosed spaces or when the lead plates contain arsenic, sufficient arsine may be generated to cause serious intoxication. Such cases have occurred in submarines.

Physiological action. Arsine unites with hemoglobin, destroying the corpuscles and causing an intense hemolysis. The symptoms depend in part upon the high grade of anemia so produced, and in part upon the effects produced by the elimination of the dissolved corpuscles. Accordingly the symptoms begin with malaise, great weakness, fainting fits or collapse, and these are followed by hemoglobinuria, icterus, painful liver and a variable degree of nephritis. In bad cases vomiting sets in early, with intense pain in the back, and collapse followed by convulsions, delirium and coma. The urine contains hemoglobin, albumen and casts. There may be suppression of urine. Death is caused by anoxemia resulting from the destruction of the blood and by the acute nephritis. In mild cases the symptoms are vomiting, weakness and dyspnea on exertion, anemia, jaundice, and hemoglobinuria. The red cells may drop to two million.

Treatment. There is no specific treatment except to remove the patient from the gas. Other treatment is symptomatic and such as would be used to relieve any case of acute nephritis. The hemoglobinuria ceases in several days, but the jaundice may remain a week or more. In certain cases arsenical peripheral neuritis appears after other symptoms have cleared up. In cases not rapidly fatal the prognosis is good, recovery usually being complete in a month or six weeks. The gas mask affords protection against arsine.

NITROUS FUMES (NO_2)

Nitrous fumes are given off in considerable quantity whenever smokeless powder, cordite or other high explosives are burned. They are not produced in toxic quantities when such materials are

exploded. These fumes are particularly dangerous because they are not irritating and may cause but slight irritation of the eyes and a little coughing. Yet if exposure to these fumes is prolonged, some four to eighteen hours later acute pulmonary edema develops and progresses rapidly, death often occurring within a few hours. In respect to this latent period, followed by serious acute pulmonary edema, these fumes act much like phosgene. Such cases have occurred several times on ships when powder has been burned. The irritant effect of these fumes is probably due to the formation of nitric acid by combination with the moisture in the respiratory passages. The absorption of this nitric acid in the depths of the lungs produces an injury very similar to that caused by phosgene which liberates hydrochloric acid under similar conditions (see Chapter XVI).

Prophylaxis. The gas mask will protect against these fumes, and should be worn whenever men are exposed to them.

Treatment. If pulmonary edema should develop, the treatment is precisely the same as has already been described for the pulmonary edema developing after phosgene poisoning.

OZONE

The fact that long continued inhalation of pure oxygen may be irritant to the lungs was mentioned in Chapter VII. Ozone (O_3) is even more toxic. Concentrations of 5 to 10 per million (0.009 to 0.019 mgm./liter), inhaled for a short time, produce in man increased amplitude of the pulse, drowsiness and persistent headache. Prolonged exposure to concentrations below one part per million does not produce noticeable disturbance but with higher concentrations there is irritation of the respiratory tract leading to fatal pulmonary edema (3).

Ozone is not suitable for use as a war gas owing to the fact that it changes very rapidly into oxygen. This same property makes it useful as an oxidizing deodorant and bactericidal agent. It has been used in purification of water. An atmosphere containing ozone, in any quantity, should not be entered, since the mask is no protection. Doors and windows should be opened or the ozone otherwise removed before men are permitted to enter. Ozone is readily identified by its peculiar odor.

SCREENING SMOKES

The use of smoke clouds for screening purposes is increasing. Smoke is produced for this purpose by burning smoke candles, by grenades, and by Stokes mortar and other shell. One of the best smokes is phosphorus pentoxide produced by the burning of white phosphorus. Tin, silicon and titanium have also been used usually as tetrachlorides. The commonest smoke candle in use by the American Army is the so-called B.M. candle which when burned produces zinc chloride ($ZnCl_2$).

These smokes are only mentioned in order to state that they are all non-toxic. At the same time they should be viewed with suspicion since the enemy may use this non-toxic smoke screen in order to conceal a really serious gas attack.

INCENDIARIES

White phosphorus. Phosphorus is a poor incendiary because although a great deal of heat is produced it burns from the surface gradually. However phosphorus is sometimes used in large bombs for their effect on morale. The explosion of such a bomb, scattering lumps of burning phosphorus over a large area is very terrifying and may give rise to some serious burns, since phosphorus even when embedded in the flesh will continue to burn so long as air can reach it.

Treatment. First aid treatment is directed toward stopping the phosphorus from burning. The wound should be covered with water, sterile when possible. The wound may be filled with water from a canteen. Phosphorus melts at 112°F., so that if the part is immersed in warm water, the phosphorus will melt and can be removed under water, or wiped off with a gauze sponge held in forceps. The later treatment is the same as for any other burn.

THERMITE

Thermite bombs have also been used as incendiaries. Thermite consists of 76 parts of oxide of iron (Fe_3O_4) and 24 parts of powdered aluminum. The reaction which follows when this is ignited, is $3\ Fe_3O_4+8Al=4Al_2O_3+9\ Fe$, and produces a temperature of about 3000°C. This heat melts the iron produced and this melted iron is scattered about. The explosion of shell or bombs filled with thermite is very terrifying, but actually very few casualties were caused by

themite during the war. When such burns do occur, the melted iron should be chilled at once with water. The burns produced by this or other incendiary materials are treated precisely as other heat burns.

REFERENCES

(1) SOLLMAN: A Manual of Pharmacology, Second Edition, 1922, p. 719, W. B. Saunders Co.
(2) HAGGARD AND HENDERSON: The treatment of carbon monoxide poisoning. Jour. Amer. Med. Assoc., 1921, vol. 77, p. 1065; 1922, vol. 79, p. 1137.
(3) SOLLMAN: Manual of Pharmacology, p. 618.

CHAPTER XII

Individual Protection

Theoretically absolute protection against gas can be provided; but this is seldom attained under field conditions because the equipment required would at times limit the effectiveness of the soldier disproportionately, and because of the ignorance and lack of discipline of many soldiers. All protective apparatus must be designed to give the maximum protection while complying with the following requirements:

1. Simplicity of design and function. Apparatus must be easily understood and operated even by men of small intelligence.
2. It must be comfortable and light in weight.
3. The least possible interference with the vision and mobility of the soldier.
4. A construction so strong as to be serviceable indefinitely under field conditions.

The degree of protection actually secured varies with the chemical agent used. The mask affords almost perfect protection against gases that affect the respiratory tract and eyes. It is not always possible to afford such complete protection against gases, like mustard, that also affect the skin.

THE DEVELOPMENT OF THE GAS MASK

When the Germans first used gas on April 22, 1915, the Allied troops were entirely unprotected, and nearly all of them were gassed, with the production of casualties variously estimated at from 5000 to 8000. The men who escaped with their lives did so by improvising some protection. Some buried their faces in moist earth; others applied wet handkerchiefs or shirts to their faces. In some cases men wet these cloths with urine which was probably more effective than plain water in stopping chlorine.

At first the Allies did not know what gas had been used but Professor Haldane went to France and established the fact that the gas was chlorine. Also on April 28 the first sample of a German respirator was received at the British General Headquarters. It

consisted of a gauze bag containing a pad of cotton waste soaked in a solution of sodium hyposulphite and sodium carbonate. Arrangements were at once made to have similar pads made by the women of England, and these were in use about May 4. The pads were kept in a box, to prevent drying, and at a gas alarm every man ran to the box and put a pad in his mouth. As he seldom got the same pad twice, this method was neither esthetic nor popular.

The black veil respirator. The first English production that can be dignified by the name of mask was a four fold piece of black veiling, about a yard long and eight inches wide. In the center pocket formed by the folds was placed a pad of cotton waste that had been wrung out in a solution consisting of sodium hyposulphite 100 parts, sodium carbonate 40 parts, glycerine 15 parts, and water 100 parts. The glycerine kept the respirator moist, thus obviating the necessity of dipping before use, and it was supplied in a waterproof case to prevent drying or wetting by rain. By May 15 over 200,000 veil respirators had been supplied, and they were arriving at a rate of 60,000 daily. In use, the pad was held over the nose and mouth by tying the veiling behind the head, and this mask was used by many of the British troops during gas attacks launched by the Germans on May 10 and again on May 24. This mask was chemically effective, but did not fit the face accurately and gas leaked down the side of the nose. Also many of the troops did not know how to use even this simple device, and often wet it thus making it almost impervious to air or losing most of the chemicals when it was wrung out.

The hypo helmet. This helmet consisted of a flannel sack soaked in the same solution as that used for the black veil respirator, with windows of transparent material. To wear this helmet, it was pulled well down over the head, and the lower end was tucked inside the blouse. Thus it was necessary to unbutton the blouse and rebutton it with the helmet stuffed inside, making a tight fit to prevent entrance of gas. Mica eye pieces were first used but these were found to be too fragile and later celluloid was used. By June 4, the supply of these helmets reached 10,000 per day. This helmet was a great improvement over the veil respirator, but its many disadvantages are obvious. There was a dead air space in which CO_2 collected, it was hot, and often leaked through broken windows, needle holes, or because of an imperfect fit under the blouse.

The P. helmet. In July, 1915, the British intelligence department learned that the Germans were preparing to use phosgene and the P. helmet was devised as a protection against this new gas. It was quite similar to the hypo helmet except that it had glass eye pieces and was provided with an outlet valve and mouth piece. The flannel was soaked in a solution of sodium phenolate and glycerine. This protected against 300 parts of phosgene per million, and was also a more efficient protection against lachrymators. By the middle of November, 1915, the issue of one P. helmet per officer and man had been completed. All were therefore protected against phosgene at the time of the German cloud gas attack of December 19, 1915.

The P.H. helmet. From January 20, 1916, onwards all helmets were impregnated with both sodium phenolate and hexamethylenetetramine. This increased the protection to 1000 parts of phosgene per million. The P.H. helmet was fitted with special rubber sponge goggles early in 1916 to complete the protection against lachrymators. These helmets gave efficient protection when properly worn but were subject to the following serious defects:

1. The solution frequently burned the faces of the men.
2. They were hot and stuffy.
3. They were incapable of development against other toxic gases that might be used.

In the meantime an entirely different principle had been worked out experimentally, namely, the absorption of gases by granules of lime and charcoal. Accordingly in February, 1916, the first type of box respirator appeared. It was too heavy and cumbersome for use, and the small box respirator, S.B.R., was made and investigated in April, 1916, one year after the first gas attack by the Germans. The S.B.R. consisted of a canvas satchel divided into two compartments. A canister filled with absorbent, which at first was 40 per cent wood charcoal and 60 per cent soda lime granules, was in the right compartment. A corrugated tube joined the canister and the elbow tube to which was fastened the fabric of the face piece. The elbow tube was also fastened to a rubber mouth piece which fitted between the teeth and the lips, with two lugs gripped by the teeth. The face piece was made of rubberized material, and was held in place by elastic bands which fitted over the head. Eye pieces permitted the wearer to see, and nose clips closed the nostrils permitting breathing only through the mouth.

The elbow tube was provided with an outlet flutter valve, which permitted air to be exhaled without passing through the canister. The inlet valve at the bottom of the canister permitted air to enter, but closed completely if the pressure were reversed.

The manufacture of the British S.B.R. on a commercial scale was commenced in June, 1916, and by September 19, 1916, 172,600 had been supplied to the British second army. The issue to all British Armies was completed by the end of January 1917. Thus it was almost two years after the introduction of chemical warfare before a really efficient protection was issued. Even this mask possessed serious defects. The nose clip was painful, the mouth piece uncomfortable, and saliva dripped from the flutter valve in a stream. The eye pieces were fragile and became fogged.

French masks. The French mask passed through a somewhat similar evolution. The early protection was obtained from a mask identified as M_2. This consisted of a number of layers of muslin impregnated with chemical agents. The mask was provided with goggles, and fitted the face closely so that the air could only be inhaled through the impregnated fabric. Later the French used the principle of the box respirator but developed a different type of face piece.

The Tissot mask. This mask resembled the British S.B.R. in that it consisted of a canister, tubing and rubber face piece, but differed in that mouth piece and nose clip were lacking. The face piece fitted the face closely, being held by straps about the head. Also the inhaled air enters the mask from two tubes that open directly under the eye piece and allow the air to sweep over the eye pieces. This prevents the condensation of moisture, and hence fogging of eye pieces was greatly reduced. This mask was comfortable, it was possible to speak, and salivation was eliminated. The American masks furnished during the war were of the British type, with mouth piece and nose clip, but in later models the Tissot principle was adopted.

German masks. The earliest German masks were simply pad respirators. The pads were replaced by a mask of absorbent cloth, shaped like a snout, fitting over the mouth and nose. The latest type of German respirator came into use in the fall of 1915. It consisted of a face piece of leather which was made soft and pliable by treating with oil, and fitted the face closely. A small round canister which contained the chemicals was screwed directly into

the face piece. There was neither nose clip, mouth piece or valves. The canister contained three layers, one of kieselguhr soaked in a solution of potassium carbonate, and covered with powdered charcoal, one layer of charcoal, and a layer of crushed pumice stone mixed with hexamethylenetetramine. In about April, 1918, as a protection against diphenylchlorarsine and other smokes, extensions were issued to German troops which consisted of a perforated metal cover which fitted over the bottom of the canister, being held in place by three spring clips. The cover contained a disc of porous paper, which served as a filter for the particles of smoke. The German mask is believed to be greatly inferior to both the British mask and our own war mask, but even so late as 1923 the Germans themselves claimed its superiority.

At the entrance of the United States into the war three types of mask were available: The P.H. helmet, the British S.B.R. and the French M_2 masks. Experiments were made on all three of these types, and it was soon found that the S.B.R. offered the greatest possibilities, both as regards immediate protection and future development. During the eighteen months which were devoted to improvement of the American mask, the face piece underwent a gradual evolution and the canister passed through types A to L, with many special modifications for experimental purposes (1).

The failure of the first 20,000 American masks made was mentioned in Chapter I. The American troops first used the British S.B.R., 100,000 of which were purchased in the summer of 1917. By October, 1917, the American manufacture was well established and troops were rapidly supplied with the American S.B.R. This mask was quite superior from the viewpoint of protection afforded, but the nose clips and mouth piece were so uncomfortable that action was slowed down, and after a few hours wear men would prefer to take their chances of being gassed rather than to continue wearing the mask.

In October, 1918, manufacture of the American Tissot mask was commenced. This has, with some changes and improvements, resulted in the present day type of mask.

DESCRIPTION OF THE PRESENT GAS MASK (1919, MARK 3 CANISTER, 1920 SIDE CARRIER)

The mask consists essentially of the canister, to which is connected a corrugated tube and the face piece. The air enters through the canister where it is purified. In the case of smoke and other toxic

solids, removal is accomplished mechanically by passing the air through a filter. In the case of gases purification is effected by means of chemicals contained in the canister. These chemicals consist essentially of charcoal and soda lime. These materials must be prepared so as to fulfill the following conditions:

1. A high rate of absorption. A man exercising violently may breathe 60 liters a minute and since he is inhaling only half the time, the air may pass through the canister during inhalation at the rate of 120 liters per minute. This would correspond to an average air velocity of 80 cm. per second through the canister, and since any given portion of air will remain in contact with the absorbent material only about one-tenth of a second, the gas must be completely removed during this short interval. The present mask easily removes 5000 parts per million of certain gases.

2. Absorptive capacity. The absorbent must be able to absorb and hold large amounts of gas per unit weight of absorbent. This has been accomplished to a surprising extent. All canisters used by gas casualties in the last war were tested, and in none were the chemicals broken down. The present canister affords very much more protection than did the war canister. It will undoubtedly last through any ordinary campaign. Spare canisters are always furnished to replace those broken or lost.

3. Low breathing resistance. Men exercising actively must be able to breathe freely. The present canister offers very little resistance to breathing. This is accomplished by providing materials that can be granulated and packed so as to be freely permeable to the air. This in turn increases the necessity for materials of great activity in removing the gas.

Charcoal is used because no other substance has ever been found that is as satisfactory as an absorbent of gases. The soda-lime is used because it has a very high capacity for neutralizing acid gases, like phosgene.

The canister is a tin box having three holes at the top for the entrance and exit of air. Rubber check valves are inserted in the two incoming air passages to prevent the exit of air through these openings and they are further covered by a metal rain shield. The exit for the purified air is in the center of the top between the two air entrances and is prolonged into a nozzle.

The corrugated tube is attached at one end to this nozzle and at the other end to the face piece. The corrugations in the tube pre-

vent it from collapsing and shutting off the air when it is bent or twisted.

The face piece is so made that it fits tightly to the face of the wearer, and permits no air to enter except that which has passed through the canister. The face piece is made from a special rubber compound whose outer surface is covered with stockinette, and is held in place by an elastic harness which fits around the head. Glass plates in metal frames are inserted opposite the eyes. An aluminum angle tube is inserted in the front of the face piece. This angle tube contains two passages. The upper passage is for the entrance of the purified air from the canister. A rubber deflector on the inside of the face piece causes the dry incoming air to pass up across the inner surface of the eye pieces thus preventing condensation of moisture which would seriously obscure vision. The lower passage is for the exit of the exhaled air and to its outer extremity is attached a rubber flutter valve which prevents the entrance of air through this passage. The face piece is made in several sizes in order to fit a large or small face. The sizes run from 1 to 5, one being the smallest. Masks are manufactured with face pieces in the following proportion: 50 per cent twos, 35 per cent threes, 10 per cent ones, 4 per cent fours, 1 per cent fives.

The mask is contained in a canvas satchel which is carried under the left arm where it is held in place by a sling of webbing which passes over the right shoulder and supports the weight, while a waist strap of webbing holds it close to the body. The opening is in front and is closed by a side flap with snap fasteners. The satchel can thus be opened by a single movement of the left hand and the face piece pulled out with the right.

The canister is carried in a compartment in the extreme rear of the satchel, and two horizontal straps of webbing hold the canister in the proper position. The upper strap has a snap fastener at one end, which when unfastened permits the removal of the canister from the satchel. The forward compartment of the satchel is larger and carries in the bottom the corrugated tubing and above this the face piece. The flap cover is so made that when the mask is adjusted to the face, the cover can be passed over and around the corrugated tubing and fastened again. This closes the satchel when the mask is in use, and the canister is thus protected from rain and other injurious agencies.

This mask fits the face closely, and gives complete protection to the respiratory passages and eyes. It is light, weighing 5 pounds, 2 ounces, and is comfortable. It interfers very little with the movement or vision of the soldier. It is simple in construction, and there are no parts that will easily get out of order, and no adjustments to be made by the wearer.

Two types of canister are issued, the green and the olive drab. The green canister is for training purposes only. The olive drab canister is the war canister.

While the two canisters appear to be quite different, the principles of construction of these and all other war canisters are practically the same. Canisters in general consist of the tin containers, which vary greatly in design; a mechanical filter, to remove toxic smokes and particulate chemicals; and a granular gas absorbent mixture. The gas absorbent used is a mixture of 60 per cent activated charcoal and 40 per cent soda-lime. The activated charcoal is made by taking charcoal made from cocoanut shells and heating it in a Dressler tunnel kiln in the presence of water vapor to a temperature of 850°C. for four hours. This process removes all the volatile hydrocarbons from the fine capillaries of the charcoal, leaving in each granule an enormous capillary bed which has the property of absorbing many gases. The gas is "adsorbed" to a large extent, that is to say the molecules of the gas are strongly attracted by the carbon and are held firmly bound against the walls of the capillary bed. Many of these gases can later be removed by placing the carbon in a vacuum, and the extent to which a carbon sample holds on to its adsorbed gas is spoken of as its "retentivity." The adsorptive activity of charcoal varies with the temperature, being much less at higher temperatures.

In addition to its adsorptive action charcoal acts catalytically upon a number of reactions to which war gases are subject. An instance is its action upon the hydrolysis of phosgene into hydrochloric acid and carbon dioxide, and its slight catalysis of arsine. In fact it appears that charcoal has a pronounced chemical action on most war gases. This chemical activity is affected by the water content of the gas-air mixture, phosgene being more rapidly broken down in the presence of moisture. On the other hand the adsorptive power of carbon for certain gases such as cyanogen chloride is reduced by the presence of moisture in the air. But as the charcoal

soon gives up its excess moisture no deterioration of the canister content is to be feared from any other than an actual wetting.

While charcoal possesses remarkably high absorptive powers against most gases, it suffers from certain defects. It has too little capacity for certain volatile and highly acid gases such as hydrocyanic acid and phosgene and moreover does not hold them firmly, there being a tendency for such gases to be slowly given off to a current of air. Certain other gases are best handled by oxidation and this cannot be done with charcoal alone. To make up these deficiencies 40 per cent of the canister filling consists of granules of an alkaline oxidizing agent called soda-lime. There are many formulas for various soda-limes; a typical soda-lime is prepared as follows:

Composition of wet mix

	per cent
Hydrated lime	45
Cement	14
Kieselguhr	6
Sodium hydroxide	1
Water	approximately 33

The above mass is spread in slabs, allowed to dry and then ground to 8 to 14 mesh, the resulting granules are sprayed with a strong solution of sodium permanganate and again dried. The final granule has a moisture content of 13 per cent and a sodium permanganate content of 3 per cent.

The functions of the various components of the granule are as follows:

Hydrated lime. This provides the bulk of the chemical action of the granule, neutralizing acid vapors, especially those given off by phosgene during its catalysis by the charcoal.

Cement. This is merely a binder and adds hardness to the granules.

Kieselguhr. The addition of cement decreases porosity and the introduction of kieselguhr restores this porosity and increases the absorbent surface.

Sodium hydroxide. This is added for two purposes: First, to rapidly neutralize acid gases which might not be caught by the more slowly acting hydrated lime, second, to maintain by its hygroscopic properties the water content of the granule and so prevent undue drying or deliquescence.

Sodium permanganate. This is added to oxidize certain gases such as arsine, which are not well absorbed by charcoal or plain soda-lime granules.

The above paragraphs briefly describe the components of a typical canister filling. When it is realized that the inspired air in passing through the canister is only in contact with the absorbent for one tenth of a second and that in this time the canister contents must be capable of reducing the concentration of any gas from say, 1000 p.p.m. to 1 p.p.m. or less, the necessity for carefully prepared absorbents is plain.

The present canister filling admirably fulfills the theoretical requirements for the ideal absorbent, which are

1. Absorptive activity—a rapid and complete reduction of concentration.

2. Absorptive capacity—long life in the canister under field conditions owing to difficulty of replacement. Also there must be a firm and permanent binding of the absorbed toxic gas.

3. Versatility—the filling must be of such a nature that it can be relied upon to protect against any kind of toxic gas.

4. Mechanical strength—the granules must be hard and not break down to dust under rough handling of the canister.

5. Chemical stability—the absorbents must not inter-react chemically or with the inspired air, CO_2 and moisture.

6. Low breathing resistance—the size of the granules and depth of layer must be such as to provide the least resistance to air passage, and to provide the required degree of protection.

7. Ease of manufacture, cheapness and availability of raw materials.

The mechanical filters used to remove toxic smokes have consisted of various substances none of which have been perfectly satisfactory. Cotton pads, layers of filter paper, "doughnuts" of tissue paper and a host of others have been tried and found wanting. A felt filter is excellent but the difficulty of manufacture and high cost are a disadvantage. It is believed that filters do not depend upon the capillary openings being smaller than the smoke particles for their filtering effect. Filtration seems to result from diffusion of the particles to the walls of the capillaries brought about (1) by the Brownian movement of the smoke particles (2) the area and arrangement of the internal surface of the filter, (3) the flow of the smoke as a whole, and (4) the attractive forces between the filter

surfaces and the smoke particles. The fourth factor determines how firmly bound the particles will be by the filter.

The green canister (type J) consists of an elongated oval can with corrugated sides. The air enters through a hole in the bottom, protected by a rubber check valve. A dome shaped wire screen in the bottom of the canister keeps the contents from falling out. The total volume of the charcoal and soda-lime filling is 300 cc., packed in two layers, two-thirds in the bottom and one-third in the top. A smoke removing pad is placed between the two layers and another smoke removing pad on top of the upper layer. A wire screen and spring in the upper part of the canister serve to keep the filling pressed down and in place. The purified air goes to the corrugated tube of the face piece by way of a metal tube passing through the top of the canister.

The O.D. canister is of quite different design. Owing to the introduction of finer filters a marked increase in pressure drop with resultant increase in the difficulty of breathing occurred and it became necessary to enlarge the filtering surface. The O.D. canister has an interior metal container, the sides of which are pierced by numerous small holes like a strainer. In this canister is placed the usual absorbent mixture packed around a central wedge-shaped tube extending about two-thirds the length of the can. The wedge is enlarged at the top and made circular where it passes through the top of the can to connect with the corrugated tube. The wedge-shaped tube is made of perforated tin and is covered with thin cloth to prevent dust from the chemicals passing into the tube and into the lungs. The outer side of the larger perforated tin (that containing the absorbent) is covered with felt, stamped in at the top and bottom. In reality the above is a complete canister in itself but in order to protect against weather and wear it is placed in the outer O.D. can. This outer can has two rubber check valves which admit the air through the top. Over these two valves is a metal covering to protect them from rain or from being struck or torn. Thus the air, entering the two openings at the top circulates around the loosely fitting outside corrugated case to all parts of the filter and after passing through the filter continues through the perforations of the tin into the charcoal and soda-lime and then into the central wedge-shaped tube which leads it to the corrugated tube and the face piece.

Caution. Although this canister protects against all war gases, it does not protect against carbon monoxide, hydrocyanic acid and ammonia. For use in such atmospheres special canisters have been devised. Naturally no canister can protect against a lack of oxygen. Atmospheres containing no oxygen can only be entered with oxygen breathing apparatus, or gas mask face piece with a long hose attached leading out to pure air.

TRAINING AND GAS MASK DRILL

It is necessary that all troops be drilled in the use of the gas mask. Gas is usually a surprise attack, and most casualties occur because the mask is not put on rapidly and correctly, or because it is removed too soon. Gas attacks are especially effective against poorly trained troops, and there is a great difference between troops having good gas discipline and those which have not. The commanding officer of each unit is responsible for the proper training and instruction of his command in defense against gas. It is especially important that the men of the Medical Department should be well drilled and disciplined. Many of them will be at the front with first line troops or engaged as litter bearers, and will almost certainly be exposed to gas attacks. For the most part they will be scattered among other troops, and often will be acting without any officer to direct them. Each man must therefore be trained to such an extent that he can act in the matter of gas on his own initiative when this becomes necessary. The drill must be so thoroughly mastered that all will protect themselves instantly and automatically on hearing gas alarm or when gas is noticed.

Drill A—To sling the mask. (*1*) Sling, (*2*) Mask. At the command (*1*) grasp the metal hook above the two snap fasteners with the left hand at the same time grasping the metal clasp at the extremity of the shoulder sling with the right hand. Hold the mask waist high in front of the body with side containing snap fasteners next to the body. At the command (*2*) extend the left arm sideways to full length. At the same time pass the shoulder sling over the right shoulder with the right hand, then bring the two hands together across the chest and fasten the hook and clasp together. Adjust the satchel in its proper position under the left arm. Pass the waist strap around the waist and fasten together in front.

Drill B—Drill by numbers to obtain accurate adjustment of the face piece. Proficiency in this drill will be followed by practice

without the numbers to insure as quick an adjustment as possible without sacrificing accuracy. Practice in holding the breath will also be included. The drill must be practiced until a complete and accurate adjustment can be made quickly by all.

The head piece may be worn either with the strap adjusted under the chin or at the back of the head. A modification in the drill is necessary according to the method used.

First method, the head piece adjusted with a strap under the chin, (*1*) By the numbers, (*2*) Gas. Stop breathing. Place rifle (if unslung) between knees. With left hand open flap, with the right hand knock off head piece from behind and, going on down with the same movement, bring the right arm in front of the body; keeping the forearm at right angles to the upper arm, until the hand is on a level with the opening of the satchel. Thrust the right hand into the satchel, grasping face piece between the thumb and fingers just above the angle tube. (*2*) Bring face piece smartly out of satchel to height of chin, holding it firmly in both hands with the fingers extended and joined outside of the face piece, the thumbs inside at the binding midway between the two lower straps of the head harness. Thrust out the chin. (*3*) Bring the face piece forward digging the chin into it. With same motion guide straps of the harness over the head with the thumbs. (*4*) Feel around the edge to make sure the face piece is well seated. See that head harness is correctly adjusted. (*5*) Close outlet valve by pinching between thumb and fingers of right hand to prevent passage of air through it, and blow vigorously into the mask, completely emptying the lungs. (*6*) Pass the flap of the satchel around the corrugated tube and fasten on the outer snap fastener. Replace head piece.

Second method, the head piece adjusted with strap at the back of the head. (*1*) By the numbers, (*2*) Gas. Stop breathing. Place rifle (if unslung) between knees. With left hand open flap, at the same time holding the right arm with forearm across the front of the body, the forearm at right angles to the upper arm and at the same level as the opening of the satchel. Thrust the right hand into the satchel, grasping face piece between the thumb and fingers just above the angle tube. (*2*) Bring face piece smartly out of satchel to height of chin, holding it firmly in both hands with the fingers extended and joined outside of the face piece, the thumbs inside at the binding, midway between the two lower straps of the head harness. Thrust out the chin. (*3*) Bring face piece forward,

To Sling the Mask. First Position

To Sling the Mask. Second Position

To Sling the Mask. Third Position

Mask Drill. Helmet with Strap Under Chin. One
Opening flap and knocking off helmet

MASK DRILL. ONE CONTINUED

Bring right hand down to level of opening, keeping forearm at right angles to upper arm.

MASK DRILL. TWO

Taking the face-piece out of the satchel showing the proper method of grasping the face-piece.

MASK DRILL. TWO CONTINUED

Bringing up face-piece to level of face; chin thrust out preparatory to putting on mask.

MASK DRILL. THREE

Digging the chin into face-piece, and drawing the elastic harness over the head.

MASK DRILL. FIVE

Clearing the face-piece of gas by closing the outlet valve with the right hand, and breathing out. The exhaled air passes out around the edges of the face-piece.

MASK DRILL. SIX

Passing the flap of the satchel around the corrugated tube and fastening on outer strap fastener.

MASK DRILL. HELMET WITH STRAP BACK OF HEAD. ONE

MASK DRILL. TWO

MASK DRILL. THREE

MASK DRILL. TEST FOR GAS

MASK DRILL. REMOVE MASK

MASK DRILL. REMOVE MASK CONTINUED

Showing proper method of folding face-piece preparatory to placing in satchel.

digging the chin into it. With same motion guide straps of the harness over the head with the thumbs, knocking the head piece off backward. (*4*) Feel around the edge to make sure the face piece is well seated. See that head harness is correctly adjusted. (*5*) Close outlet valve by pinching between the thumb and fingers of right hand to prevent the passage of air through it, and blow vigorously into mask completely emptying the lungs. (*6*) Pass the flap of the stachel around the corrugated tube and fasten on the outer snap fastener. Replace head piece.

Note: A lanyard may be attached to the left loop of the head piece, and the other end is passed around the left shoulder. The purpose of this is to prevent the head piece from being lost when knocked off during the adjustment of the mask.

Drill C—Method of testing for presence of gas. Mask being adjusted (*1*) Test for gas. Take a moderately full breath. Stoop down so as to bring the face close to the ground, but do not kneel. Insert two fingers of right hand under face piece at right cheek. Pull the face piece slightly away from the right cheek and sniff gently. If gas is smelled, readjust the face piece. Close outlet valve by pinching between thumb and fingers of right hand and breathe out hard, thus clearing the face piece of gas.

Drill D—To remove the mask. Having tested and found no gas: (*1*) Remove, (*2*) Mask. Remove head piece and hold in left hand. Insert the first two fingers of right hand under face piece at the chin with the thumb on the outlet valve guard. Bend the head forward, at the same time removing the face piece with an upward motion of the right hand. Replace head piece.

Note: Care must be taken to remove the mask without undue stretching of the elastic.

Drill E—To replace the mask. (*1*) Replace, (*2*) Mask. Grasp face piece in right hand with edges of face piece turned upward, the fingers under the left eye piece and thumb under right eye piece. With the left hand place harness pad inside face piece just above eye piece. Unfasten flap. Slide the corrugated tube into the bottom of the satchel until the angle tube has passed the satchel entrance, then turn the edges of the face piece toward the back of the satchel and push the face piece into the upper empty part of the satchel

above the corrugated tube. Fasten flap using inner snap fastener on upper corner of flap.

Note: After all drills, the eye pieces should be rubbed with antidim, leaving a thin, transparent film of the composition on the glass, the face piece should be wiped dry, folded correctly and put away in such manner that the outlet valve is not bent.

DISINFECTION OF MASKS

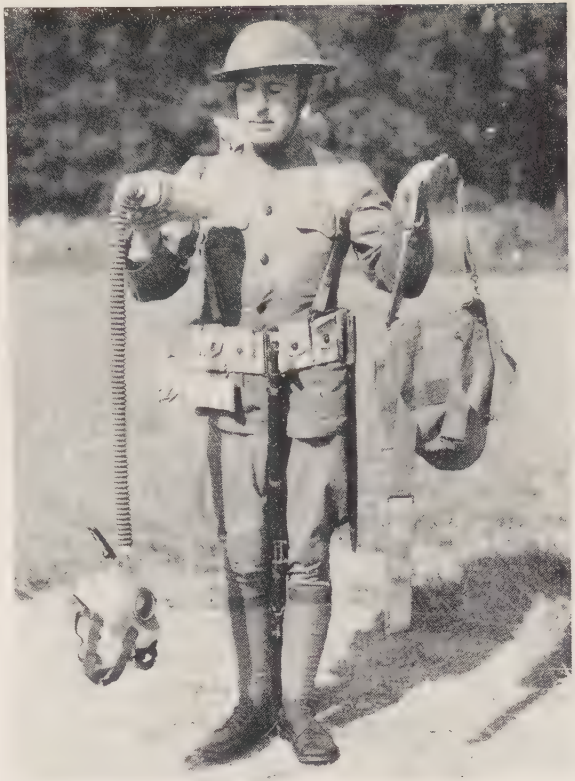
Former models of the mask necessitated breathing through a mouth piece, and the entire mask became soiled with saliva. Elaborate instructions were therefore issued from time to time for the disinfection of these masks especially when they were used by more than one man for training purposes. When using the present model, there is no soiling of the face piece, and it is improbable that disease will be transmitted by it. If it is felt that disinfection is required, the inside of the face piece may be wiped out with a cloth saturated in a 2 per cent solution of liquor cresolis compound. The moistened part should be thoroughly dried before returning it to the satchel.

CARE OF THE MASK

Keep the mask from all unnecessary moisture and dry it out if wet. Carry nothing but the mask in the satchel. Keep the mask where it cannot be damaged when not in use, and inspect all masks at regular intervals.

Drill A—To prepare for mask inspection. (*1*) Prepare for mask inspection. Place rifle (if unslung) between knees. Unsling mask. Open flap of satchel and take out complete mask including canister. Hold satchel in left hand and canister in right hand.

Drill B—Mask inspection by the numbers. Being prepared for mask inspection, (*1*) by the numbers, (*2*) Inspection, (*3*) Mask. Free right hand by holding canister in left armpit the tube and face piece hanging over upper left arm. Examine exterior of satchel and sling making sure that sling is properly fastened to satchel, that all metal hooks, clasps, buckles, snap fasteners, and D. rings are present and are fastened securely in place, that satchel has no holes or tears and that stitching is not ripped. Examine interior of satchel. See that the two straps which hold the canister in place in the rear compartment, are present and not damaged or ripped loose, that the snap fastener for the upper strap is complete and not loose



MASK INSPECTION DRILL. "PREPARE FOR INSPECTION"

INSPECTION MASK. ONE

INSPECTION MASK. TWO

INSPECTION MASK. THREE

INSPECTION MASK. FOUR

INSPECTION MASK. FIVE

INSPECTION MASK. SIX

INSPECTION MASK. SEVEN

including both the part on the strap and the part on the side wall, and that the can of anti-dim is present in the loop of the lower strap. (*2*) Fasten the hook and clasp of the shouldersling together. Slip the left arm through the sling and allow the satchel to hang from the upper left arm, at the same time removing the mask therefrom by grasping the canister with the right hand. Examine the canister for rust spots and weak places by pressing lightly with the fingers beginning at the bottom and working toward the top. Look under rainshield and see that check valves are present. See that rainshield is not loose. See that canister contents do not rattle on shaking. (*3*) Adjust the mask to the face, making sure of a proper fit. Then pinch together the walls of the corrugated tubing just above the canister nozzle and try to inspire air. If air cannot be inspired, the mask is free from leaks. If a leak is detected, its approximate location may be determined as follows. Pinch the walls of the corrugated tube together at the angle tube. If leak is no longer detected on inspiration, the leak is in the corrugated tube, otherwise it is elsewhere. If the leak is found not to be in the corrugated tube, then pinch together the flutter valves at the angle tube, and also the corrugated tube. If leak is no longer detected on inspiration then the leak is in the flutter valves, otherwise it must be in the face piece by process of elimination. Having approximately determined the location of the leak, or having determined the absence of leaks, proceed with the minute inspection according to the remainder of the drill. Examine corrugated tube for obvious tears, punctures, or other defects. See that it is properly connected to the canister nozzle and to the angle tube, noting in this connection that the adhesive tape over the binding wires is present and in good condition. (*4*) Examine the flutter valve for tears and pinholes, by distending the rubber between the fingers. Look especially for pinholes just below where flutter valve is joined to angle tube and for tears around valve opening. See that valve has no dirt or sand in it and that it is properly connected to the angle tube. See that the binding wire is properly taped. See that flutter valve guard is not loose. (*5*) Examine outside of face piece for tears or other damage to stockinette. See that angle tube is properly connected to face piece with rubber band surrounding the binding. See that the fabric has not torn or pulled loose around the eye piece frames. Examine the chin seam and see that it is in good condition and properly taped inside and outside. Examine

the inside of the face piece for pinholes by creasing the rubber inner surface backward thus causing pinholes to appear due to their enlargement from stretching. See that the deflector is in good condition properly connected to the angle tube and properly cemented to the sides of the face piece. Test the entire face piece for softness and pliability. (*6*) Examine the head harness. Make sure that it is complete, that the straps are firmly attached to the face piece, to the harness pad, and to each other as the case may be. See that the adjustment buckles are in good condition and that the straps have not lost their elasticity. (*7*) All men with defective masks step forward one pace. Others replace mask in the satchel taking care to replace canister and face piece in proper position.

Notes: (*a*) After mask inspection the men should be given Mask Adjustment. Drill once so that it can be readily seen whether all masks have been correctly replaced in the satchel.

(*b*) Failure to detect a leak under (*3*) does not mean that mask is free from defects. This procedure indicates actual leaks only, and does not indicate impending leaks or other defects caused by rotting tape and rubber, rusting wire, pinholes which only partially penetrate the fabric, etc. It is therefore necessary to complete the drill whether leaks are detected under (*3*) or not.

GAS MASK FOR HEAD INJURY CASES

If the head or face is wounded, it becomes impossible to wear the regulation mask previously described; and unless a special gas mask is available for such cases, they must submit to gassing in addition to their injury. The Germans recognized this necessity and made a special gas mask for these cases. Such a mask should possess the following qualifications:

1. It should afford as complete protection as the regulation mask.
2. It must be capable of application over large surgical dressings, to an unconscious man.
3. It should be of one size, yet must fit the largest or the smallest man.

Mr. Randolph Monroe of Edgewood Arsenal has designed such a mask in collaboration with the Medical Research Division. This mask has a sack shaped hood about 36 inches in circumference, and provided with elastic tapes and buckles by means of which the sack may be taken up after the hood is adjusted. This prevents the possibility of dead air space. The closed head end contains the usual non-shattering glass eye pieces, and angle tube, flutter valve,

corrugated tubing and canister as in the regulation mask. The canister is attached by webbing belts to the skirt of the coat, or may lie free on the stretcher. The hood is prolonged into a coat, the bottom end of which may be fastened to the skin by a drawstring, after pulling up the shirt. The only serious objection to this mask is that it will undoubtedly be hot in warm weather. However it will only be necessary to wear it during the time required to transport the patient beyond the gassed area. Specimens of this mask are to be stored at Edgewood Arsenal, to serve as models for the manufacture of such numbers of this special mask as may be required by the emergency.

PROTECTION FOR THE SKIN. PROTECTIVE CLOTHING

The first development of protective clothing was for the purpose of securing protection for operatives in mustard plants. The first suit developed provided protection for the entire body. The suit was a single garment with tie strings at wrists and ankles, and the head was protected by an aluminum helmet. Such a suit would be practically useless under field conditions. Later another type of impervious overall suit was developed, which protected against mustard gas for over one hundred minutes. This suit was made of cotton material impregnated with linseed oil containing a non-drying material. These suits were very uncomfortable, especially in warm weather, because they entirely prevented the escape of perspiration from the body. This, of course, will be the main objection against any variety of impermeable material. Rubber clothing is of no value because rubber is a good solvent of mustard.

Such impermeable suits were subsequently distributed among field troops where protection against mustard was needed. In brief it may be said that such suits are quite impracticable for fighting men, although they serve an excellent purpose for wear for short periods by men employed in extra hazardous employments, such as by men detailed to destroy liquid mustard splashes.

The Chemical Warfare Service has since developed compounds known as Impregnite. These compounds possess the property of destroying the activity of mustard. Ordinary clothing may be impregnated with these compounds. Clothing so impregnated is capable of neutralizing mustard for several hundred hours under field conditions. Such clothing is non-irritant, can be laundered without destroying its protective power, but is increased in weight about 15 per cent.

By wearing such impregnated underwear and outer uniform, complete protection against mustard vapors is afforded for all parts of the body except the hands, feet and head.

It has been found possible to treat leather gloves and shoes with another compound, which enables these articles to resist mustard vapors indefinitely, and liquid mustard for several hours. An impregnated hood pulled over the head would complete the protection against mustard.

It will thus be seen that, theoretically at least, complete protection against mustard can be afforded. This has not been tried out under field conditions. But men have actually worn this clothing for several months without suffering any inconvenience, and it seems probable that in future it will be possible to equip troops with mustard-proof clothes without in any way hampering their mobility or interfering with military objectives. Thus a great tactical advantage will be secured since one of the most important tactical uses of mustard is to render ground unfit for the passage of troops.

PROTECTION FOR HORSES

Protection for horses is not as necessary as for men, yet cavalry may have to traverse a gassed area, and draft horses may be used in hauling supplies and ammunition. Horses are practically unaffected by lachrymators, and they can stand much higher concentrations of pulmonary irritants than can men. But the horses' skin and hoofs are very susceptible to mustard. If horses are taken through a mustard area without protection, most of them will be casualties. The Germans, French and British all developed various types of gas mask for horses. The American type finally adopted is a cheese-cloth bag covering the nostrils and upper jaw. Horses do not breathe through the mouth and it is unnecessary to cover the lower jaw. The mask has a canvas mouth piece which prevents the cheese-cloth from being bitten through. The bag is provided with a drawstring which passes around the opening and enables the mask to be drawn tightly about the face. The ends of the string are sewed on to elastic bands which form the mask head harness. These elastic bands keep a tension on the drawstring when the mask is adjusted. Inside the cheese cloth bag, and attached to the canvas mouth piece is a canvas frame which is stitched to the bag in such a way as to prevent the bag being drawn into the nostrils when in use. In breathing, the horse must draw air through

this cheese cloth bag which is impregnated. The chemical employed consisted of a mixture of hexamethylenetetramine (protection against phosgene), nickel sulphate (protection against HCN), sodium carbonate and glycerine. Horses can run two miles with this mask without showing evidence of exhaustion.

Theoretically this mask gives good protection. Practically it was not a great success. It is easy to put this mask on a horse once as he probably thinks it is a new type of feed bag. But later, horses fight against the mask so that it is difficult to adjust at best. In the confusion of a night attack, with the drivers themselves wearing masks, it becomes doubly difficult.

Horse boots are probably of more value than the mask. Mustard was used more than any other gas in the later months of the war, and mustard attacks the horses' hoofs producing inflammation, especially around the coronary band. A special hoof pad and boot was devised.

The pad was made of sheet iron imbedded in a hoof protector of composition rubber to which the shoe was applied. The shoe just overlaps the metal plate on the inside, and provides a solid metal surface for the bottom of the foot. The boot was made of satin treated so as to be impervious to mustard gas. It covers all the foot except the bottom and extends to just below the knee. The boot is held in contact with the hoof by a sewed cloth strap which passes around the bottom of the hoof, and is held in position by projections extending from the spur or toe clip. Special care is taken to insure a perfect joint at the rear of the boot, since the small cavity in the back of the hoof is one of the most sensitive parts. The boot is wrapped about one and a half times around the leg and is clipped with five loops through which passes a $\frac{3}{4}$-inch strap (1).

REFERENCES

(1) Fries and West: Chemical Warfare. McGraw Hill Book Company, 1921.
(2) Official History of the War, vol. II, 1923, p. 334.

CHAPTER XIII

Collective Protection

The term collective protection is self explanatory. It includes all those measures which are difficult or impossible of execution by an individual, but which become available to field units after proper organization and training.

ORGANIZATION OF GAS DEFENSE

An officer of the Chemical Warfare Service is assigned to the staff of the Division Commander. His duties are to act as an adviser in the preparation of plans of offense and defense.

Unit Gas Officers and N.C.O's. The training of troops in Individual and Collective Protection, and in general protective precedure, is carried out under the supervision of the Chemical Warfare Service by officers and non-commissioned officers detailed from their own organizations for this purpose. Every regiment appoints a Regimental Gas Officer and Non-commissioned Officer; each battalion of the regiment appoints a Battalion Gas Officer and Non-commissioned Officer; and each company appoints two Company Gas Non-commissioned Officers. Other separate units also appoint for the same purpose, at least one Unit Gas Officer and Non-commissioned Officer. The term Unit Gas Officer and Non-commissioned Officer is a general one which refers to all of the officers and non-commissioned officers described above, without reference to the particular organization to which they belong. Unit Gas Officers and Non-commissioned Officers receive special training to fit them for their duties from the Chemical Warfare Service, which exercises technical supervision over their work in the field, although they work, of course, under orders from the commanding officers of their respective units. During active operations against an enemy, Unit Gas Officers and Non-commissioned Officers should be relieved from other duties.

The work of the Unit Gas Officers differs from that of Chemical Warfare Officers in that it is mainly limited to Defense Against Gas. It should be stated in this connection that they also perform most important work in the domain of Chemical Warfare Intelligence, and

are the principal and most dependable sources of chemical warfare information obtained through field channels. Their primary function, however, is to prevent gas casualties in their respective units, and their intelligence work is mostly in connection with this end in view. It is the duty of the Division (or corresponding higher unit) Chemical Warfare Officers to render these officers all possible assistance and guidance in their work.

Independent medical organizations similarly should appoint a gas officer and suitable non-commissioned gas officers whose duties should be the training of the personnel in individual protection, and the enforcement of all measures that will afford collective protection for his unit against gas. Thus the gas officer of a field hospital would be charged with the duty of preventing casualties among the personnel of the hospital and its patients, and may also become responsible for the care and treatment of gas casualties should such occur.

METHODS FOR FORETELLING THE PROBABLE USE OF GAS

Gas is most likely to be used when conditions are favorable. These favorable conditions depend chiefly upon weather, terrain, and time.

1. Weather. The velocity of the wind is of prime importance. A high wind increase the dilution of gas enormously and rapidly dissipates it. Non-persistent agents such as phosgene are comparatively ineffective in a wind over 12 miles an hour. On the other hand persistent agents such as mustard are not so much affected by wind velocity. When the ground is saturated with liquid mustard, a high wind may dissipate the gas, but enough of the liquid remains to be effective when the wind drops. If cloud gas is to be used, a moderate velocity such as 8 miles an hour is to be desired. For if the velocity is low, the direction shifts rapidly, and the gas may be blown back upon those using it or away from the target. A low velocity is favorable to the use of all gas except cloud gas. Wind direction is of prime importance. Cloud gas need not be expected unless the wind blows more or less directly from the enemy. Again should the wind be blowing toward the enemy, it is improbable that gas will be received on the front lines, although troops further back may be readily gassed by shell containing non-persistent gases. Mustard would not be used by the enemy when the wind is blowing toward him, except when it could be placed far enough back to prevent any of the gas from returning. For this reason, reserve troops are more likely to receive mustard gas than those in the front line.

Warm, sunny days make non-persistent gases less effective. For, on a warm day, the temperature of the earth is higher than that of the air and ascending air currents are formed which carry the gas up, and also disperse it. On the other hand a hot day is favorable to the use of the persistent gases such as mustard, as the liquid is vaporized more readily, and much higher concentrations may be secured. Cold weather is unfavorable to the use of the mustard type because volatilization is reduced. Mustard freezes at 13.9°C., and at this temperature is practically useless. Cold weather does not affect the non-persistent gases nearly so much.

Cool cloudy weather may in general, be regarded as favorable to the use of cloud gas, and hot weather for mustard. Rain is distinctly unfavorable as it washes the vapor out of the air and in many cases causes hydrolysis of the compound as well as destroying persistent agents on the ground. Therefore although gas might be used in a light rainfall, it is improbable that it would be used during a heavy rain.

It is obvious that proper meteorological observations will be of value not only in indicating the most favorable time for using gas against the enemy, but also to indicate when gas is most likely to be used against us.

2. Terrain. Tall grass, bushes, trees, buildings, etc., retard the movements of air and gas, thereby making it more persistent. In dugouts, cellars, buildings, shell holes, deep hollows and in woods, gases persist longer than in the open. In light winds, the contour of the terrain is the controlling factor of the wind direction. A valley whose main direction deviates less than 45 degrees from the direction of the wind, will usually control the course of air currents and gas clouds. The effects of gas from projectiles will be diminished in marshy ground.

3. Time. Night is the most favorable time for a gas attack for two reasons. Meteorological conditions are always more favorable at night because ascending air currents caused by insolation are absent. Surprise is the greatest element in the success of gas attacks, and there is always more probability of surprising sleeping men. Therefore special vigilance is required at night and all sleeping men must be guarded by gas sentries.

Season is of some importance. It is obvious that mustard or other liquid agents that freeze cannot be used during the winter. Moreover each season generally has its prevailing winds which may be advantageous or the reverse in that particular locality.

4. Other indications of an impending gas attack. The installation of Livens projectors and particularly of cylinders for cloud gas attacks involves an enormous amount of labor and a considerable increase of personnel in that particular sector. Increased activity within the enemy lines should be noted, and air plane photographs may show the installation of apparatus. When it is known from such observations that a gas attack is impending in any sector, the attack may be expected when weather conditions are favorable. Unfavorable conditions however do not preclude the possibility of attack, which may be made at such times in order to increase the element of surprise.

SPREADING THE GAS ALARM

After a gas attack has been launched there is usually an interval before the gas reaches the target. It is often possible to detect the attack immediately and give warning during this interval.

1. Cloud gas. During the day the jets from the cylinders can be seen, and when the humidity is high, the cloud itself can be seen some distance away. Also the first portion of the cloud that reaches the target is of low concentration as compared with the main body of the cloud that will follow in a few seconds. (See Travel of Gas and Smoke Clouds.) As soon as the odor is detected by advance sentries the alarm can be given to all troops over which the cloud will pass. This is evidently especially necessary at night when sleeping men must be awakened to put on masks.

2. Artillery gas shell. It nearly always happens that one or two projectiles burst in advance of the others, thus giving a warning of gas that may be communicated to others, before any heavy concentration of gas can be set up by this method.

3. Projectors. The simultaneous discharge of a number of projectors produces a very loud detonation, the sound of which reaches the target considerably in advance of the projectiles. There is also a very large and bright flash, which may give warning at night. However the flash may be hidden from sight by intervening hills in the terrain, and the detonation of the projectors is often masked by firing them during the course of an ordinary artillery bombardment. It is the difficulty of avoiding surprise by projector attacks and of warning men who are sleeping or busily engaged, combined with the sudden high concentrations of gas set up which make this method of liberating gas so effective. Moreover large bombs used by the air service filled with gases of the phosgene type cannot be detected in

advance. The only protection against such sudden inundations of gas is a perfect dicipline and training of the individual soldiers in applying the mask.

4. Gas sentries. Duties and qualification. All sentries act as gas sentries in addition to their other duties. Special gas sentries are also posted where needed. The most important function of a gas sentry is to enable men, by his presence, to sleep and rest without having to depend on themselves to detect the presence of gas Gas sentries should be posted over all men sleeping anywhere within the theater of operations. There must be a sufficient number of sentries to insure that every sleeping man shall be wakened in time Each gas sentry should be equipped with adequate alarm devices, should have a definite area to alarm, in case of a gas attack, and, above all, should know where every man in his area is sleeping. In the event of a gas attack, the gas sentry will adjust his mask, sound the alarm, and immediately proceed to wake up every man in his area. In order to prevent the spread of false alarms the gas sentry will give the alarm only when he, himself, detects the presence of gas, and will not pass on alarms arising in other areas.

Gas sentries should be given special training in recognizing the conditions favorable for a gas attack and they must also be thoroughly familiar with the odors of chemical agents. It requires good judgment and mental alertness to make an efficient gas sentry. The gas sentry should be impressed with the vital nature of his job, and the importance of prompt detection of a gas attack and spreading of the alarm. At the same time he should also be impressed with the serious menace caused by false gas alarms. It should be explained to him that the enemy may often try to accomplish this by sending over a few scattered gas projectiles which do not warrant an alarm. He should be taught to exercise judgment in distinguishing between a genuine gas attack, and an attempt by the enemy to undermine gas discipline.

In addition to these duties all gas sentries are responsible that the standing orders for defense against gas, as far as applies to the carrying and wearing of gas masks, keeping out of gassed areas, etc., are complied with, and that all cases of infringements of these orders are promptly reported.

5. The alarm There are two types of alarm to be given in case of an enemy gas attack; general alarms, and local alarms. General alarms are given only in case of cloud gas attacks of considerable

magnitude which are expected to involve a large area. They are sent out by the usual methods of communication, including the telephone and radio. Local alarms are given in all other cases by special alarm devices. These all depend upon the production of a raucous noise. The most widely used of these devices is the Klaxon horn, but iron triangles, empty shell cases, watchmen's rattles and various sirens have all been used. These alarms are installed in appropriate places or are carried by gas sentries. As soon as the sentry is convinced that an actual gas attack has commenced, he gives the alarm by this device. This is the signal to make all preparations for a gas attack.

METHODS OF DEFENSE

Include construction and use of gas proof shelters, the removal of gas from surroundings and clothing, and care of material.

1. Construction and use of gas proof shelters. A gas proof shelter is any enclosed space which has been so constructed that gas cannot enter it. It may be a dugout, a hut, a tent, or even a tank. Such shelters provide a place where men who have to work for long periods in a gassed area can take off their masks and rest. Gas proof shelters will enable medical aid stations to function without interruption during a gas attack without that reduction in efficiency that follows the wearing of the mask, and will furnish places where wounded men who cannot wear the mask can be placed pending evacuation.

In locating gas proof shelters care should be taken to take advantage of any natural protection. They should be sheltered from wind, remembering that high hills and deep valleys deflect gas out of the direction of the wind. By proper choice of location it may be possible to establish dressing stations where there is little chance of encountering dangerous concentrations of gas. The slope of a hill offers an excellent opportunity. Here the station may be sheltered from artillery fire, from the wind, and may be at such an elevation as to be easily reached by the wounded, and yet be above the heavy concentrations of gas, which hug the ground fairly closely.

Gas proof shelters may be constructed in accordance with two different principles: (1) An air tight construction is used and provision made that when gas is present all openings to the tent or building can be made impervious to gas. (2) A positive pressure is maintained in the enclosed space by means of air pumped in from the outside through a special canister that removes the gas. As a result of

this positive pressure air is constantly escaping through the cracks of the building or other shelter thus preventing the entrance of gas. This latter method is suitable on ships to protect men in gun turrets and other compartments. It may also be used to make tanks or other shelters that cannot be made air tight, gas proof. A gas proof shelter should be provided with a special type of gas proof entrance, especially when it is constructed on the air tight principle. This may consist of double doors reinforced with blankets, which form a close contact with slanting door frames, when they are dropped. Whenever possible the entrance should be a walled-in passage way or tunnel, extending several feet beyond the walls of the shelter, and the ends may be slanted so as to receive the door frames at the proper angle. When the entrance to the shelter is a simple horizontal passage the two doors should be placed about ten feet apart, or sufficient room to enable litter bearers to enter with a litter. If the entrance is a dugout or cellar, the entrance may be a stairway, and doors should be placed at the top and bottom. When both doors are closed, an air lock is formed. To enter the shelter the outer door is opened and the person enters the air lock and closes the outer door behind him before opening the inner door. When leaving the shelter, the reverse procedure is followed. One door must always be closed.

Tightly fitting doors may be improvised by various methods. The door frames may be made of 1-inch boards, 6 inches wide, upon which 6-inch strips of blanket may be tacked. The door should be made of 1-inch boards, and a blanket is tacked over its inner side. These blanket strips may be chemically treated with sodium thiosulphate solution or with impregnite B to destroy mustard, or may simply be kept wet. When the door is held tightly shut by a spring or other device, contact is made between the wet blanket on the outer side of the door frame and the wet blanket on the inner side of the door, making a joint impervious to air or gas.

A roll blanket door may be made. The blanket itself is cut to about 4 inches longer and wider than the door frame. The top end of the blanket is nailed by a cleat to the top of the door frame. Several additional slats, cut to the same width as the door frame are nailed horizontally at intervals across the outside of the blanket. These should be reinforced by being nailed to similar slats on the inside of the blanket. The inside slats must be two inches shorter than the width of the opening of the door frame. Weights of about

an ounce a piece are attached to the blanket from top to bottom, close to the edge, and outside that part of the blanket that falls on the door frame. When the blanket is lowered, the weights hang over the edge of the frame forming a flap. The slats hold the blanket down against the door frame, and an extra length of blanket lies on the ground. Such a door is fairly gas tight. To open such a door it is rolled up, and the roll placed upon a shelf above the top of the frame.

Tents may be made gas proof by treating the canvas with an impervious coating. Earth may be heaped over the bottom of the tent, and a special air lock entrance constructed.

To prepare a gas proof shelter for use, close both doors after wetting the blankets, plug all chimneys, windows, ventilators, etc., and put out all fires. A fire causes a draught of air up the chimney and this causes a suction in the shelter drawing in contaminated air. The fire also uses up the oxygen in a room that is already devoid of ventilation. Chloride of lime may be spread on the ground at the entrance of the shelter to destroy any mustard on the shoes of those entering the shelter.

The positive pressure protection against gas can only be used by special apparatus, namely an air pump attached to a large specially prepared canister or a battery of the canisters used in the ordinary gas masks. The air must be pumped in at a sufficient rate to maintain a slight positive pressure inside the shelter. All cracks at windows and other exits must be reduced to a minimum to avoid the necessity of pumping in an enormous volume of air. The pressure required will not be great in order to prevent gas from entering the few apertures that cannot be closed. If we assume a maximum wind velocity of 25 miles an hour and a total area open to the wind of a few square inches, the necessary pressure within the shelter will not be greater than eight pounds per square foot. Under all ordinary circumstances a much lower pressure would suffice. When first installed such an apparatus should be carefully tested out to determine its sufficiency for that particular shelter. After that it need only be operated when gas is actually present. It is probable that by an apparatus of this kind, armored tanks may be made gas proof thus enabling fighting men in them to dispense with masks and greatly heightening their efficiency. A modified, armored, gas proof tank, would be an ideal equipment for an advanced dressing station, combining as it does protection against gas, all firearms except large caliber shell, with a remarkable mobility. With these appliances it may be possible to

pick up wounded at the firing line, and proceed with them directly to the collecting station or even to the field hospital.

In general gas proof shelters suffer from two serious disadvantages. They are in the nature of semi-permanent construction and will seldom be available during mobile warfare although they were highly effective during the stationary trench warfare of the last war. The tanks mentioned above form a very important exception, as they are decidedly mobile, and can keep up with any troops. Secondly when men, who have been in a gassed area, enter a gas proof shelter, they nearly always carry gas in on their clothing. This is not so apt to occur or so harmful when it does occur, while non-persistent agents of the phosgene type are used. But toward the end of the war, mustard was the most effective gas, and even small amounts of mustard carried on clothing into a small non-ventilated room, would be sufficient to cause a dangerous concentration. Thus it would become necessary for medical units to have a shelter containing two compartments with gas proof doors between them. All patients from the gassed area entering the first compartment would remove their clothes before entering the second compartment where the doctors, nurses and other patients who function permanently in the shelter would remain. Such a system probably cannot be used beyond the point at which degassing units can be safely sent. For if clothes are to be removed, we must also provide some method of removing gas from the clothes.

2. Removal of gas from surroundings. When gas has penetrated a dugout or gas proof shelter, the best method of cleaning it out quickly after the gas attack has passed and the outside air is free of gas, is to light a fire near the inner end of one of the entrances, or in the center if there is a chimney. The draught carries the gas out.

It would be obviously impractical to carry about large quantities of supplies to neutralize gases that are non-persistent. Hence measures for the chemical neutralization of gas are confined to persistent agents of the mustard type. Even in this case it is impractical to attempt to neutralize mustard when it is spread over large areas. It is, however, important to remove mustard from important points, such as headquarters, Signal stations, the various aid stations and important roads and crossings. Chloride of lime is used for this purpose and may be sprinkled over any area when it is desirable to neutralize mustard. Each unit should have a definitely organized detail under a gas non-commissioned officer to perform this work.

The men should be equipped with protective clothing, shoes and gloves to protect them while operating in the gassed area.

In neutralizing a mustard gas shell hole, the upturned earth and ground in the vicinity are covered with a layer of fresh chloride of lime. This in turn is covered with earth. Chloride of lime should not be thrown upon liquid mustard, because the reaction that occurs generates considerable heat and vaporizes the mustard. Liquid mustard should first be sprinkled with sand or earth to absorb the larger part, after which it should be well covered by chloride of lime. Leaky shells that have failed to explode should be buried, surrounding and covering the shell with a thick layer of chloride of lime.

When in a mustard area, the entrances to all dugouts, gas proof shelters, dressing stations, etc., should be well sprinkled with chloride of lime in order to neutralize the mustard on the shoes of persons entering.

3. Removal of gas from clothing. Clothes splashed with liquid mustard can be renovated by boiling for two or three hours or by steaming for an hour or more. Both of these methods are open to objection. Boiling injures woolen clothes and sterilization in a disinfector injures clothing splashed with mustard because too much hydrochloric acid is generated in the clothes from the hydrolysis of the mustard.

Another method is a follows: The clothing is placed in methyl alcohol at a temperature of 60°C. or 140°F. for five minutes. Hot water (80°C. or 176°F.) is then run into the bath bringing the volume to five or six times the original amount. The liquid is then run off and the clothes washed in cold running water for thirty minutes. In this method the mustard is dissolved out of the clothes by the alcohol, and hydrolyzed when mixed with the water.

The best method of treatment is to immerse the clothes in a solution made as follows:

	per cent
Turkey red oil (sulphonated castor oil)	5
Sodium bicarbonate	4
or	
Laundry soap	5
Water q.s. ad	100

The clothing is left in this solution at 20°C. for one hour or at 80°C. for fifteen minutes, then thoroughly washed in plain water and dried in the open air.

Turkey red oil is an article of commerce, readily obtained. Since it is an oil, it dissolves mustard freely, and being sulphonated it also dissolves in the water in which the mustard is hydrolyzed. Practically all clothing whether made of cotton, wool or leather, may be treated by this method without injury.

Should none of these methods be applicable, it will probably be necessary to burn heavily contaminated clothing.

Chlorine may be used to neutralize the mustard on slightly contaminated clothing. The men enter a gassing chamber containing one part of chlorine to a thousand of air (2.86 mgm. per liter) wearing their gas masks. After leaving the chamber the clothing should be collected and washed, the men bathed and supplied with fresh clothing. This is affected by Mobile bathing units.

Mobile bathing units. These have been adopted as field equipment. Two units should be supplied each division, and should be under the control of the Division Surgeon who may send them where most needed.

The unit consists of (1) one motorized bathing truck, (2) one 3 ton truck of standard make for transporting clothing and equipment, (3) one motorcycle with side car for the commanding officer, (4) one 50-foot ward tent for cover.

The bathing truck is equipped with a 1200 gallon tank, a rotary pump for filling the tank and a device for heating the water and generating steam. The bathing section consists of twenty-four shower heads, each capable of ejecting 4 quarts of water per minute. They are attached to $1\frac{1}{2}$-inch tubing which is arranged in three independent section frames. Each section frame is 10 feet long and is attached to the hose leading from the boiler. The showers are supported upon a portable iron upright made of 2-inch tubing with sliding joints at a height of 7 feet 6 inches above the ground.

Demonstrations have shown that this entire unit may be set up within seventeen minutes from the time it arrives on the ground. Only two minutes are required to set up the shower baths alone without the tent. Twenty-four men can be bathed in one minute and a half, and with a water supply there is no limit to the number of men that can be bathed. 700 men can be bathed with the water transported on the bathing truck.

The equipment for the unit, carried on a 3 ton truck consists of the following: One ambulance guidon on lance staff, 1 flag (blue), 4 axe heads, 6 axe helves, 4 spades, 6 lanterns (two with blue globes), 3

pick axes, 4 litters with slings, 1 hospital ward tent, 1 portable shower apparatus with 24 shower heads, 2 Vermorel sprayers, 2 barrels of lime, 4 small oxygen tanks, 4 Haldane masks, sterilizing water bag, 4 galvanized iron cans, 1 box of soap, bicarbonate of soda, and clothing.

Clothing to be carried with the bathing unit. After a careful study of the records in the clothing department of the Quartermaster Corps it was found that the great mass of clothing worn by our troops was confined to a few sizes which could be consolidated into four issue sizes. It was also realized that clothing could not be issued in separate articles and that the same would have to be assembled into units of issue. As a result, all clothing was arranged in sizes as follows: for soldiers 62 to 66 inches, size 1; 66 to 68 inches, size 2; 68 to 70 inches, size 3; 70 to 72 inches, size 4. A few extra sizes were also carried for emergency. The unit equipment for each man consisted of one suit of underclothing, flannel shirt, breeches and blouse, all being assembled and securely fastened in a single kit, ten of which were packed in canvas bags with the size plainly marked on the outside. When applying for clothing the men would give their height, and a kit consisting of unit equipment would be handed them. This system proved very satisfactory.

Amount of clothing with each unit. The following amount of clothing in gas proof bags, was carried with each unit, 500 blouses, assorted sizes; 500 breeches, assorted sizes; 500 flannel shirts, assorted sizes; 500 undershirts, assorted sizes; 500 underdrawers, assorted sizes; 500 pairs puttees (woolen leggings); 500 hand towels.

Organization and distribution of personnel of bathing unit. One Captain or First Lieutenant, Medical Corps—general supervision, reports, records and returns; 1 Sergeant, First Class, Medical Department, in general charge of records and clothing; 1 Sergeant, Medical Department, in charge of bathing section; 1 Sergeant, Medical Department, Chief chauffeur and mechanic; 7 privates, first class or privates, Medical Department, 1 Private, first class, to be chauffeur.

Departments of the bathing units. The bathing units are divided into sections as follows:

a. Reception Department, for the receiving of all men to be bathed.
b. Bathing and Irrigation Department.
c. Clothing and Evacuation Department.

When the unit was operating, the following system was observed. All men reporting to the unit were carefully scrutinized for vesicant

symptoms. Those presenting these symptoms were treated immediately and evacuated, the others divided into sections or squads of 24 men each for the purpose of systematizing the bathing. The members of these sections then stacked arms, removed all loose equipment and laid it on the ground in front of them. They then proceeded to the lime box, where their hands were disinfected with dry lime, the same method being used for disinfecting their shoes. Blouses were then removed and disposed of according to directions, following which they proceeded to the Reception Department in a tent and took position in front of their designated numbers on the canvas where they were instructed in the manner of taking baths. Prior to entering the tent, helmets were dipped in a tank of lime water for the purpose of degassing.

Method employed in disrobing. In disrobing, the following system was adhered to:

a. Helmets were placed on the numbered canvas sections in front of the men and used as receptacles for holding valuables, small trinkets, etc.

b. Flannel shirts were then removed and disposed of according to directions.

c. Shoes and socks were then removed and placed with the other retained equipment.

d. The underclothing was then removed and placed in the proper receptacles.

While men were disrobing, they were not permitted to sit down, but remained standing, thus avoiding all possibility of infecting the places which were to be used later when rearranging their clothing.

After all members of the squad were disrobed and their clothing and equipment properly disposed of, they marched in column of files to the bathing department, where they took their places under the shower heads. Before the showers were turned on, the men sprayed themselves with liquid soap. At a given time the showers were turned on, allowing fifteen seconds for wetting the bodies. When time permitted, three-fourths of a minute was allowed for scrubbing the body, following which the showers were again turned on for thirty seconds. After the bath, clean towels were issued and, after being used, they were placed in proper receptacles. Passing from the showers, the men's eyes, noses and throats were sprayed with a solution of bicarbonate of soda, 5 per cent strength. They then proceeded to the kit department, where they received clothing kits returning to their places and quickly readjusting their clothing.

While stripped, the men were again examined by medical officers, for vesicant symptoms. Those exhibiting genuine signs were immediately evacuated to the hospital. After the men redressed, they returned to their commands.

In order to avoid delays in waiting, sections or squads of 24 men were ready to proceed to the baths as soon as they were vacated.

The bathing units were designated during the daytime by the displaying of a large blue flag, at night by a blue lantern.

CARE OF SUPPLIES

Many supplies are injured by the various gases used. Many of them have a corrosive action on metals, and a phosgene or chlorine cloud may put machine guns out of action because of corrosion of the working parts, particularly of the ammunition. Similarly all surgical instruments and metallic medical supplies may be seriously damaged. Most chemical agents also render food and water unfit for consumption.

Whenever possible all such supplies should be protected during a gas attack by storage in gas proof shelters. Articles that are in constant use, such as surgical instruments, may be well oiled after using and kept in special gas-proof containers. Boxes well lined with tar paper or oil cloth with tightly fitting tops or doors will serve the purpose. Instruments in pouches should be well wrapped in oiled paper. Immediately after the attack, all unprotected instruments or metallic supplies should be thoroughly washed, polished and oiled. If soiled with mustard, protective gloves should be used, and all rags used in cleaning should be burned in the open or buried.

Food may be kept in similar boxes rendered gas proof by careful lining and covering with oil cloth. In cases of great emergency, food not so protected may be used after exposure by cutting off all the outside portions. However it is far safer to discard all food that has been exposed to gas; water so exposed should always be discarded. Men should be particularly warned against drinking or bathing in any water exposed to possible contamination. Many men were burned bathing in the water of shell holes that contained mustard, and mustard as well as all of the arsenical compounds such as D.A. and D.M. are poisonous when taken internally.

DRAFTING INSTRUCTIONS FOR DEFENSE AGAINST GAS

Standing orders for defense against gas should include all routine measures of protection. The following points should be covered in such an order,

1. Commanding officers of all units are responsible for the training of their respective commands in methods of defense against gas; for the maintainance in perfect condition of all protective apparatus; for taking the proper protective measures before, during and after a gas attack.

2. The appointment of unit gas officers and non-commissioned officers, together with relief from other conflicting duties.

3. The time to be spent and nature of the training in defense against gas should be specified.

4. A zone should be designated within the limits of which all protective measures are to be carried out. This is the danger zone. Owing to air service operations it may include the entire field of operations.

5. Within the danger zone every one must carry a gas mask at all times.

6. Frequent and regular inspections of all materials for protection should be required.

7. The posting of an adequate number of gas sentries, especially over all sleeping men must be required.

8. The duties of unit gas officers, non-commissioned officers and gas sentries should be outlined.

9. Commanding officers of all units should be directed to prepare plans of defense against gas whenever they expect to occupy an area more than twelve hours.

10. Individual action during a gas attack should be prescribed. This should include adjustment of mask, spreading of alarm, awakening of sleeping men, lowering of gas proof doors, putting out fires, cessation of unnecessary movement, etc.

11. The removal of the mask after a gas attack without the authority of an officer or the non-commissioned officer in charge should be forbidden.

12. The posting of sentries around an area unfit for occupation should be prescribed.

13. A definite organization should be specified for destroying mustard or other chemical agents.

14. The inspection and proper care of all material after a gas attack should be outlined.

15. Food and water that has been through a gas attack should be discarded except in cases of necessity.

Procedure prior to, during and after a gas attack. The unit gas officer must plan what his unit will do should an attack be delivered, and informs the next higher commander of this plan. As soon as his unit goes into an active sector he makes a careful study of the terrain and general circumstances and makes recommendations as to the best location to occupy. The location and extent of nearby gassed areas is determined, and an inspection made of all roads, bridges or other territory that must be crossed by his unit. Gas sentries are then posted and apparatus installed for protection.

In conformity with the above instructions, as soon as it is definitely established that a gas attack is in progress, the alarm is given and all routine measures described are carried out. The unit gas officers determine as quickly as possible the nature and extent of the attack and plans for defense are carried out, if necessary moving to a previously selected location.

If a chemical agent of the phosgene type is being used, all unnecessary movement should cease and troops in direct contact with the enemy should prepare to repel an assault. Medical organizations in the rear should use gas proof shelters if available. Should a persistent agent of the mustard type be used, since it is certain that continued exposure will produce numerous casualties, the unit should be withdrawn if possible to a gas free location. Otherwise the described precautions are taken. Casualties are removed from the gassed area as rapidly as possible.

After a gas attack the most important procedure is to be certain that all are prepared for another attack. Troops are more liable to surprise after having passed through one attack as they are prone to assume that the danger is over for the present. The unit gas officers study the attack estimating the purpose of the enemy and determining the number of casualties produced. They will make a report on the state of gas discipline in their unit, and should casualties have occurred owing to defective apparatus or discipline, will take immediate steps to correct these defects. They should determine location and extent of contaminated areas, and post sentries to prevent further occupation of these areas. All material remaining after the attack, as unexploded or leaky shells are disposed of, ground

around important posts is properly treated, and food and water inspected and destroyed or otherwise treated.

Although the procedure outlined appears very simple, experience during the past war indicated that the greater number of casualties were caused either through ignorance or wilful disobedience of these instructions. Medical officers should take especial pains to see that their units do not offend in this way.

The chief duty of a medical officer during a campaign is to maintain the greatest possible number of effectives on the firing line. In this capacity he gives instruction on sanitary matters and on gas discipline. It is especially disgraceful if his own unit is the first to violate his own instructions.

CHAPTER XIV

Field Organization of the Medical Department for Treatment and Transportation of Gassed Cases

Chemical agents are used:

1. To produce casualties
2. To harass and lower morale
3. To render strong positions untenable
4. To conceal movements, and to lead enemy to draw wrong conclusions
5. To damage enemy material and supplies

From the viewpoint of the medical officer, the important point is that surprise is the most important factor in the success of chemical warfare operations. Thus smoke may be used at one time to screen an infantry advance; on another occasion to give a false impression of such an attack, thus causing the enemy to expend ammunition uselessly, and bring up reserves thereby weakening other parts of his line. Still again, smoke may be used to screen an actual toxic gas attack. By good judgment in varying these chemical warfare agents, the enemy is kept continually guessing and thrown into confusion. Should an enemy use chemical warfare agents against us he will undoubtedly employ the same tactics. Thus no great argument need be expended in proving that medical personnel must be prepared at all times for such surprise attacks, including preparation for the treatment of wounded or gas casualties in the presence of gas.

MEDICAL ORGANIZATION FOR TREATING GAS CASUALTIES

It is practically impossible to have a definite table of organization detailing personnel at the various aid stations for the treatment of gas casualties. Any such organization must be extremely flexible, since at any particular time there may be no gas casualties while on another occasion there may be a large number. It is evident that any medical officer, whether in the zone of combat, along the line of communications, or at the base may at any time be compelled to care for gas casualties. Every officer and enlisted man of the Medical Department should have a thorough knowledge of the latest treatment of chemical warfare casualties. This knowledge will be just as neces-

sary and important to medical officers as a knowledge of the proper treatment of wounds and fractures. A knowledge of first aid for gassed cases will be far more important for the enlisted man of the hospital corps than training in the treatment of arterial hemorrhage, which after all is comparatively rare.

All of the relief stations now contemplated by the scheme of organization will probably be furnished with special equipment for the treatment of gassed cases, and additional personnel will be sent to these stations as the need arises. The relief stations to be considered in this organization are as follows:

1. Battalion and regimental aid stations
2. Collecting stations
3. Ambulance companies
4. Hospital center
5. Evacuation hospitals
6. General hospitals

BATTALION AND REGIMENTAL AID STATIONS

When occasion demands, Chemical Warfare Sections will be established in connection with battalion and regimental aid stations. The number of these sections and their size depend upon the probable requirements. As a rule, like other sections of Battalion and Regimental aid stations, they should be established as near the front lines as conditions will permit. As far as possible all such aid stations should be protected against the effect of chemical warfare agents. The need for protection against such agents increases with proximity to the front, and the difficulty in providing gas proof tents or other shelters increases likewise.

The treatment to be given gas casualties at these stations is limited to first aid as described in previous chapters, which may be summarized as follows:

1. Patients gassed with pulmonary irritants: Clothing should be loosened, and equipment removed. Wrap patient in a blanket, make him lie down, and whenever possible, evacuate seriously gassed cases on a litter in the recumbent position. Evacuation should be as prompt as possible in order that these cases may reach a hospital where they may receive effective treatment before the onset of serious pulmonary edema. Doubtful cases and those evidently not seriously gassed may walk back to the collecting station.

In this connection, the following is quoted from page 321, Official History of the War, vol. 2, London, 1923.

> Deffnite cases of gas poisoning (pulmonary irritants) are harmed by any form of transport, since this leads to unavoidable muscular exertion. On the other hand, the choice of the time for transport is often determined by military considerations, and the ultimate fate of the casualty depends on appropriate treatment, and above all, on trained nursing, which is best afforded in the gas station of a permanent hospital. An attempt should therefore be made to transfer the case from the medical dugout to a gas station within an hour or two of being gassed. If the case is only taken as far as the main dressing station, a decision has to be made whether further transport or the lack of efficient nursing constitutes the greatest danger.

2. If vesicants have been used, such parts of the patients' clothing as have been splashed with liquid mustard or lewisite should be removed. It is impractical to remove all clothing at this station for this would convert otherwise ambulant cases to transport cases. All splashes of mustard should be removed from the skin by kerosene or solution of chloride of lime as described previously. Wash eyes, nose and throat with 2 per cent bicarbonate solution.

If lewisite has been used, apply 5 per cent sodium hydroxide solution, or ferric hydrate paste as described previously.

It should be emphasized that patients that have been gassed with mustard or similar vesicant agents, should not be allowed to use transportation to the rear that is needed by wounded men or cases gassed by pulmonary irritants. About twenty-four hours will elapse before mustard cases are seriously affected, and they are therefore entirely capable of walking all the way to the collecting station or even to the hospital center. This should be required as long as transportation is needed for other cases. They should certainly never be litter cases.

On the other hand should practically all casualties be due to mustard, as may easily happen, ambulances may then be used to get these cases as rapidly as possible to the nearest aid station where they can receive a bath and change of clothes. This may be possible at the collecting station, and will certainly be possible at the hospital center.

It is obviously a general rule that if the medical officer and his personnel are obliged to wear the gas mask, patients must also be compelled to wear them until all danger is past. When the medical personnel can remove their masks, patients may do likewise.

Special supplies required at Chemical Warfare Sections of Battalion and Regimental Aid Stations

Gas proof tentage?
Spare blankets
Kerosene—in pint tins, Chloride of lime, Sodium bicarbonate } for removing mustard
Sodium hydroxide, Collapsible tubes of ferric hydrate paste } First aid treatment of lewisite burns
Morphine, Picric acid, Alcohol } Treatment of heat burns
Gauze and cotton, Olive oil } Mustard and other burns
Dichloramine-T, Chlorcosane } Mustard and other burns
Ammonia, Chloroform, Ether, Alcohol } Inhalation mixture for toxic smokes
Emetine hydrochloride-hypodermic tablets
Urease solution?
Hypodermic syringes.

COLLECTING STATIONS

Collecting stations are generally established from 1 to 2 miles back of the front line, but conditions of the terrain, shell fire and other circumstances may necessitate their establishment further to the rear. Collecting stations are therefore still within the zone of active chemical warfare, and equipment should be furnished which is proof against these agencies. It is highly undesirable to locate them in underground cellars or dugouts unless these can be made gas proof. Valleys should also be avoided because gas flows into them. Collecting stations may preferably be established on the side of a hill where they will be protected at least partially both from fire and from gas.

If hostile troops are using gases, chemical warfare sections are established by the Collecting Companies of the Medical Department. Extra personnel can be detailed to these sections from reserve medical units at the rear. All such personnel detailed to chemical warfare sections must be thoroughly instructed in the best methods of handling gas casualties. The chemical warfare sections of collecting stations may be divided into the following departments:

1. Receiving
2. Bathing
3. Departments for slightly gassed
4. Department for seriously gassed
5. Department for observation
6. Forwarding

1. Receiving department. Here the case is diagnosed, and tagged if this has not been done earlier. It may be expected that a certain number of men will attempt to escape from the front claiming to have been gassed. Others may be very slightly gassed, and others seriously gassed. Every effort should be made here to sift out the non-gassed cases and return them to the front. When there is doubt as to whether the man was gassed he should be sent to the department for observation. Slightly gassed cases should also be retained in the department for slightly gassed. By this procedure the medical department is fulfilling its first purpose in battle, the maintenance of the effective fighting force, and at the same time the clogging of the entire medical system with able-bodied or only slightly injured men is prevented.

2. Bathing. Mobile bathing units should form part of the Chemical Warfare Collecting Stations. Here cases that have been exposed to mustard or other vesicant gases should have their clothes removed, should be thoroughly washed and should be given fresh clothing. The eyes, nose and throat should be carefully washed out. Such cases may then be sent to the forwarding section.

3. Department for slightly gassed. Trivial mustard burns may be dressed with dichloramine-T in chlorocosane, and these cases will then require little further treatment and may be sent to the forwarding section. Cases gassed with pulmonary irritants should be warmly wrapped and provided with a comfortable and sheltered place where they may lie quietly. They may be given hot coffee and possibly soup. The more serious cases may also be given intravenous injections of gum glucose solution should it appear desirable. This treatment definitely delays the onset of pulmonary edema. No further treatment is necessary at this time, but all such cases should be carefully watched for the signs of beginning pulmonary edema, i.e., dyspnea, cyanosis, rapid weak pulse. If these symptoms appear, intravenous gum glucose solution should be given at once.

4. Department for seriously gassed. There will probably be very few serious mustard burns at this station, for the reason that it

requires about twenty-four hours for severe burns to develop. All mustard casualties are therefore bathed as soon as possible, and dressings applied where necessary in other sections of the collecting station described above. These cases should then be directed to walk to the hospital center or if transportation is available it may be used for prompt transportation to the hospital center or even further back, to an evacuation hospital where facilities may be at hand to care for them should serious burns develop.

The department for seriously gassed will therefore include chiefly those cases that have been gassed by pulmonary irritants, and have already developed a certain degree of pulmonary edema. In addition to the rest and warmth which is provided for these patients, prompt measures will be necessary at once if life is to be saved. These measures include intravenous gum glucose injections, or if time does not permit this treatment, bleeding for cyanosed cases, intravenous cardiac stimulants for the gray cases with failing heart action, and the administration of oxygen to both classes. Cases with failing heart action or considerable cyanosis are not capable of standing transportation and should be held here until they improve or die. All less serious cases that can bear transportation should be sent to the forwarding section for prompt evacuation to the hospital center.

5. Department for observation. This department will exist chiefly to determine whether cases have actually been gassed. All cases that do not show definite signs of gassing twenty-four hours after exposure may be returned to the front. Should the presence of mustard gas be definitely known, such cases should be bathed and held, or later sent to the department for slightly gassed. Should very slight burns have developed after twenty-four hours, such cases may be dressed and either held or returned to the front according to circumstances. The hospital center and lines of communication should not be permitted to become clogged with slight burns that obviously require little or no treatment.

6. The forwarding section sends cases received from the other sections to the field hospital by ambulance companies. Much judgment will be necessary to send the most urgent cases first. Cases that have obviously been exposed to mustard without the mask, or that have been badly burned, liquid lewisite burns, and cases who are developing pulmonary edema but are still transportable should be evacuated first. Cases for immediate transport should have this

properly indicated on their tags. Cases that will require oxygen administration on the ambulance should also be plainly marked to that effect.

Equipment required at Chemical Warfare Sections of Collecting Stations

Tents for seriously gassed cases
Blankets—extra clothing
Mobile bathing units
Dichloramine-T
Chlorcosane
Sodium bicarbonate
Chloride of lime
Alcohol
Kerosene
Emetine hydrochloride
Caffein sodio benzoate hypodermic tablets
Digitalin tablets
Sterile 300 cc. flasks of gum glucose solution
Sterilizible syringes—2 to 5 cc.
Instrument case, bleeding and minor operations
20 cc. syringes with two way stop-cock, or irrigators for intravenous gum glucose injections
Portable oxygen apparatus

AMBULANCE STATIONS

Ambulance stations are points established for the administration and control of ambulance companies. One ambulance station is usually established by a company of the ambulance battalion somewhat to the rear of each collecting station. This ambulance company is charged with the duty of transporting as rapidly as may be, all cases from the forwarding department of the collecting station, to the hospital center. All ambulance companies operating in zones where chemical warfare is used should be prepared to care for gas casualties. Little can be done for mustard burns on the ambulance. But they should in addition to their regular equipment, carry an extra supply of blankets, and should be prepared to give cardiac stimulants and to administer oxygen to cases suffering from pulmonary edema. Each ambulance should carry as many oxygen cylinders with proper face pieces (Haldane apparatus) as there are patients to be carried. When face pieces are not available, oxygen may be administered by attaching a small rubber catheter to the valve of the cylinder.

The end of the catheter is passed into the naso-pharynx of the patient, and the gas turned on. This is a relatively inefficient method of administration as compared with a proper face piece, but will be better than no treatment.

In this connection it should be mentioned that the use of armored tanks has been suggested for the transport of casualties. The largest type of armored tank, if stripped of its fighting gear, can carry 30 patients, 8 of whom could be lying on litters. It can travel over the roughest terrain, and over smooth ground can travel at a rate of 10 miles per hour. Such a tank may also be made gas proof since it can be made almost air tight, by the simple device of a small air pump which will maintain a slight positive pressure inside the tank. The air from the outside would be drawn in through a modified gas mask canister.

Such tanks could proceed directly to Battalion aid stations, pick up serious wounded or gassed cases, and could carry them to the collecting station or even directly to the Hospital Center. Even with the hospital center 5 miles behind the fighting line, such a tank could make a round trip in one hour, whereas cases transported by litter from the front to the collecting station, and thence to the hospital center by ambulance would arrive a variable number of hours later.

Over good roads, the tank can move as rapidly as the ambulance and it can proceed over terrain that ambulances cannot pass. Nor is the cost of these tanks excessive. The cost of such an ambulance tank may be placed at $50,000, only approximately twice as much as a Martin bombing plane.

If one of these ambulance tanks were assigned to each brigade, the transportation of wounded and gassed cases would be greatly facilitated.

THE HOSPITAL CENTER

The sorting sections of hospital centers were designated as Triages during the war. Probably for the same reason that many officers prefer to wear an English uniform instead of an American. These sorting sections are most important because it is here that patients are sorted out for final disposition. As a rule these sorting stations are established at the entrance to the Hospital Center about 3 to 6 miles back of the firing line and generally out of the gas zone. How-

ever, shells filled with mustard or other chemical warfare agents may be dropped here unless the hospital is sheltered from shell fire by the terrain, and cloud gas attacks in the past have at times proved effective for a depth of 10 miles.

So far as the treatment of gassed cases is concerned the duties of the field hospital are as follows:

1. Sorting section. Non-gassed or trivial cases that have filtered through the collecting station should be carefully examined and held here until a positive diagnosis can be made. This can certainly be done in from twenty-four to forty-eight hours after the alleged exposure. Non-gassed cases may then be returned to the front, and real gas casualties sent to the proper ward for treatment. All cases that are obviously suffering from the effects of gas when they arrive are hastened to the ward where they may receive proper treatment.

2. Bathing. The receiving ward should have a field shower bath apparatus as it is important for all mustard cases to receive a thorough bath. This may be the first station at which a bath can be administered, as this will only be possible at the collecting stations under exceptionally favorable circumstances. Connected with the bathing establishment should be a de-gassing station with apparatus for the removal of mustard and similar compounds from the clothing and equipment.

3. Hospital wards of the field hospital should be set aside for the treatment of the various gassed cases. If these cases arrive in considerable numbers, it may be necessary to designate an entire hospital for the exclusive care of gas casualties. At least, mustard cases should be segregated in one ward, and cases suffering from pulmonary edema in another. When possible these groups should be further broken up into groups of slightly gassed and those who are seriously ill.

The treatment of these cases will not be described here as it has been detailed in other chapters. The question at once arises how long should these cases be kept at the hospital center. This hospital is supposed to be a mobile institution and cases are ordinarily kept here only a short time.

We may therefore give the following rules for the disposition of gas casualties, bearing in mind the fact that circumstances alter cases, and that rules, like paint, must be mixed with brains if they are to be successful:

1. An effort should be made to keep all trivial gassed cases here with the expectation that after a few days they may be returned to the front.

2. Cases that have been severely gassed by mustard should be evacuated to the rear as soon as their temporary wants have been supplied. They will be casualties possibly for weeks or months, and they should be sent through the evacuation hospital to the base general hospital where they will be cared for permanently, as soon as they have received food and drink, dressings, and a night's rest which may have to be secured with morphine. There is no objection to the use of morphine in mustard casualties, though it must not be given cases suffering from pulmonary edema. Mustard does not cause acute pulmonary edema.

3. Cases suffering from pulmonary edema as the result of phosgene or similar pulmonary irritants must be kept and treated here until the height of their illness is past. This will only be a matter of forty-eight hours at the most. At the end of that time after exposure, such cases are either dead or on the road to recovery. However, they will not be fit for service for possibly several months more. Therefore as soon as practicable after forty-eight hours when the cases are improving, and the administration of oxygen is no longer necessary they may be transported *lying down* to more permanent hospitals. After several weeks in a base hospital, they may be sent to convalescent camps where by careful graduated exercises and other measures, they may completely recover health and strength and become fit for further service.

Evacuation hospitals are established on railways, whenever possible, at a distance of from 8 to 16 miles from the battle front. They provide facilities for the care of all classes of casualties and serve as centers from which evacuations from the combat zone to the communications zone are carried out. Evacuation hospitals are mobile units of the army.

Convalescent hospitals, located well to the rear of and central to the army area, receive from evacuation hospitals slight, convalescent, and other cases offering prospect of early restoration to combat fitness. Their function is to hold these cases under army control in order that they may be returned to duty at the earliest practicable date. Army convalescent hospitals usually find employment only in stabilized situations.

The evacuation of patients from the army area is effected by means of hospital trains or improvised railway transportation, by barges and other means of water transport, and, when necessary, by motor transportation (1).

*French medical war data concerning proportion of gas casualties**

Ratio of gunshot casualties to gas casualties in 1918

	MARCH TO JUNE	APRIL TO MAY	JUNE	FINAL WEEK OF THE WAR
	French troops back	Stabilized front	Open operations	French troops advance to Belgium
	per cent	*per cent*	*per cent*	*per cent*
Gas	3, 8	40	11, 17, 30	6
Gunshot	97, 92	60	89, 83, 70	94

*From The Military Surgeon, December, 1923, page 591.

CONCLUSIONS

When troops advance or retreat rapidly (open operations), gas casualties decrease in relation to gunshot casualties.

When troops offer resistance (French operations), gas casualties increase in relation to gunshot casualties.

REFERENCES

(1) Field Service Regulations, United States Army, 1923.
(2) Chemical warfare and its relation to the medical department. Army Medical Bulletin, August 1, 1923, p. 201.

CHAPTER XV

The Ultimate Results of Gas Poisoning

There has been a very general belief that the subsequent existence of those men gassed during the war was extremely pitiful and that gas poisoning caused much disability and chronic invalidism. Whether this is true or false can only be learned by examining the facts. There are two series of facts at our disposal: namely, attempts that have been made by foreign or American observers to determine the ultimate disposition and present condition of gassed cases, and observations upon the actual rate of recovery in experimental animals.

ULTIMATE DISPOSITION AND PRESENT CONDITION OF GASSED CASES

The British figures are perhaps the best for our purpose because there were a large number of gas casualties, the cases have been followed more carefully than in other countries, and the results have been published. The following figures and statements are taken from Chapter XVII of the Official History of the War, vol. II, London, 1923. The known total of British gas casualties in France is shown in the following table:

	TOTAL GAS CASUALTIES	TOTAL DEATHS
Cloud attacks with chlorine, April and May, 1915..	7,000*	350
Cloud attacks, December, 1915 to August, 1916....	4,207	1,013
"Lethal" gas shell, July, 1916 to July, 1917.....	8,806	532
Gas shell, July, 1917 to November, 1918 (mustard gas period).....	160,526	4,086
Projector attacks, December, 1917, to May, 1918...	444	81
Total.....	180,983	6,062

* Approximate figures for casualties admitted to medical units only; many casualties died on the field or were taken prisoner.

Of the surviving 174,921, many were gassed more than once thus causing a double entry, and the figure of 150,000 is accepted as a reasonable estimate of the total number surviving after gas poisoning.

According to information supplied by the Ministry of Pensions, from August, 1919, to August, 1920, the resurvey boards made 26,156 examinations of 22,000 cases of poisoning. Of this number 3136 showed no disability whatever, leaving the total number of pensioners at about 19,000, which was about 12 per cent of the total gas casualties. Gas poisoning was responsible for 2 per cent of all pensionable disabilities, 35 per cent being caused by wounds and injuries, and 63 per cent by disease. Obviously gas was responsible for only a very small percentage of the total pensionable disabilities, and it may be assumed that 88 per cent of the total gas casualties received no serious permanent injury since they made no application for pension. This also illustrates the difference between the manner in which pensions are managed in England and in this country where there was a total of 70,552 possible gas casualties and 300,000 applied for war relief alleging gas disability. This disgraceful situation is only one manifestation of the almost universal tendency to try to get something for nothing from benevolent Uncle Sam, who as is well known is both rich and foolish.

But what was the condition of the 19,000 cases that drew a pension? The following table shows the actual assessments in a consecutive group of 2416 examinations made by boards during four weeks in September, 1920.

DISABILITY ASSESSMENT	NUMBER OF CASES	PERCENTAGE OF TOTAL
100 per cent disablement	12	0.49
90 per cent disablement		
80 per cent disablement	3	0.12
70 per cent disablement	11	0.45
60 per cent disablement	12	0.49
50 per cent disablement	39	1.6
40 per cent disablement	106	4.3
30 per cent disablement	387	16.0
20 per cent disablement	840	34.7
Less than 20 per cent	720	29.8
Nil	286	11.8

From this it will be seen that the average degree of disability was about 20 per cent. Very few men were seriously or totally disabled. A severe shortness of breath from bronchitis or emphysema, and pulmonary tuberculosis were assessed 100 per cent, so that it is obvious that such serious cases were rare and that the disabilities complained of were in general comparatively trivial.

In March, 1919, the papers of 150 gas pensioners were examined. These cases were selected as being of a more serious nature than the average. The disabilities found were grouped as follows:

	per cent
Non-organic diseases:	
Neurasthenia	30
D.A.H. and debility	25
Pulmonary diseases:	
Bronchitis, asthma, etc	35
Tuberculosis	4.5
Other diseases:	
Valvular disease of the heart	4.5
Other complaints	1
	100

Valvular disease of the heart was responsible in 1919–1920 for 6 per cent of all diseases among pensioners and pulmonary tuberculosis for 5 per cent. The percentage for these two diseases is therefore about the same for gassed and non-gassed men.

Sandall, in an article entitled "The later effects of gas poisoning" in the Lancet, October 21, 1922, page 857, analyzed all the pensioners, 83 in number, in the Oxford area whose disability had been accepted by the Ministry of Pensions as due to gas poisoning.

In these cases, the symptoms complained of were as follows: (1) The principal and commonest complaint is of shortness of breath on exertion which was present in 70 per cent of the cases. (2) Cough and expectoration—usually worse in the morning and frequently worse in winter than in summer, though always persistent—54 per cent. (3) Pain, or a feeling of tightness across the chest—25 per cent. (4) Palpitation and occasional giddiness, 14 per cent. (5) Morning vomiting or nausea with want of appetite, 12 per cent. (6) Headache, 9 per cent. (7) Nervousness, insomnia, tremulousness, want of energy and other symptoms of neurasthenia, 7 per cent. (8) Lachrymation or soreness of the eyes, 5 per cent.

When we examine for physical signs to account for these symptoms we find:

Heart. Seventy-four per cent presented no abnormality whatever apart from tachycardia, which will be referred to later; by "abnormality" I imply evidence of enlargement, murmurs, or alteration in the cardiac sounds. In the 19 cases in which the heart did not appear perfectly normal 18 presented signs of slight enlargement, with the apex beat in or close to the nipple line, and 4 of these also had a cardio-respiratory systolic apical murmur; in only 1 case, however, was there definite enlargement with the apex beat outside the nipple line. Of the remaining 7 cases not perfectly normal, although there was no evidence of enlargement, 3 had an apical systolic murmur, 1 a pulmonary systolic murmur, while in 3 cases the cardiac sounds were noted, as weak and distant. It is a disputed point as to what constitutes tachycardia, but by taking a resting pulse-rate of 100 or over as evidence of this condition, we find

that 45 per cent of all cases of gas poisoning suffer from tachycardia, although not usually to a severe degree. Only 5 cases had a resting pulse-rate of over 120, 1 of 140, 1 of 150, and 1 of 160. In only 3 cases was any irregularity of action noted. The exercise response was tested in every case, whether tachycardia was present or not, and the response was good in every case where tachycardia was absent. In cases with initial tachycardia the actual response was fair—except in 2 cases, where it was poor—but I think we must admit that even with a fair response to a test exercise the actual capacity for work of a tachycardia case is somewhat below normal, and the condition in itself constitutes a disability. Unfortunately no blood examination was made in any case, so that it is impossible to say whether there was any departure from normal conditions in that respect.

Lungs. On physical examination of the chest the lungs presented no abnormal physical signs in 53 per cent; of the remainder, 26 per cent showed signs of emphysema to a slight or moderate degree, while 20 per cent in addition had some definite signs of bronchitis, but in 1 case only was the bronchitis severe. In only 1 case of the series, also, were there any physical signs suggestive of pulmonary tuberculosis, and in this case the evidence was not conclusive; there was a history of hemoptysis and subsequent sanatorium treatment, but tuberculosis had never been found in the sputum, and there were certainly no signs of active tuberculosis at the present time. I shall refer to this case later.

Digestive system. Turning to the digestive system, although a furred tongue was frequently present, no abnormal physical signs of any kind were found in the abdomen, even in those cases complaining of digestive troubles.

Nervous system. On examination, 7 per cent were found to present definite physical signs of neurasthenia, with tremors of the hands and tongue, and increased reflexes.

Other signs. Definite pharyngitis was found in 7 per cent, and slight conjunctivitis in 5 per cent. Lastly, in a small number of cases there was scarring or pigmentation of the skin, showing evidence of burns by mustard gas, but no case showed any dermatitis or any evidence of a skin disability at the present time.

Having considered the symptoms and physical signs presented by these cases of gas poisoning in their later stages it may be instructive to note the percentage assessment of the disability given by the medical boards, i.e., the the extent to which the later effects of gas poisoning are estimated to affect the working capacity of the patient.

	Percentage of disability
In 57 per cent	less than 20
In 21 per cent	20
In 10 per cent	30

One case was assessed at 40, 3 cases at 50, and 1 case at 70 per cent.

Conclusions

From the facts presented above we may draw one or two definite conclusions.

1. In the first place, whether physical signs are present or not, every one of these cases still complains of definite symptoms more than three years after the

armistice; no man in this area who has been awarded a pension for gas poisoning has as yet admitted that he has really recovered and that he now suffers from no after-effects, and it seems clear that every case still has some diminution of working capacity. The conclusion is that, if complete recovery does not take place at an early stage, the later effects of gas poisoning, if not permanent, are at any rate of very long duration, and lower the standard of health of the individual for an indefinite period.

2. In nearly half the cases (46 per cent) there is evidence of emphysema or chronic bronchitis or both. The reports of the Chemical Warfare Committee, and, in 1915, Sir J. Rose Bradford and Dr. T. R. Elliott[1] gave detailed descriptions of the pathological conditions found in the lungs in fatal cases. In addition to the damage to the mucous membrane of the air passages a severe disruptive emphysema was present, and we may conclude that in the cases now under consideration the same changes would be found in a less severe degree. These cases, therefore, are unlikely to improve, and will probably tend to get worse in later life. The disability will be permanent, treatment on the usual lines will not differ from that of cases of emphysema and bronchitis in ordinary civil life.

3. There is a popular impression that pulmonary tuberculosis is a frequent sequela of gas poisoning, but I do not think this is borne out by the facts, in this area at any rate. I admit of course that a certain number of tuberculosis cases, invalided out of the service for this cause, give a history of gas poisoning; the latter is not always substantiated, however. Gas poisoning may, of course, stimulate latent tuberculosis into activity, but it is a remarkable fact that of all the cases of gas poisoning in this area in receipt of pension not one has developed tuberculosis or is now under the supervision of the tuberculosis officer. My own feeling is that pulmonary tuberculosis is not a common effect of gas poisoning, and certainly not one of its later effects. J. B. Hawes[2] has stated that, in his opinion, many cases diagnosed as pulmonary tuberculosis after gas poisoning are not proved to be such, and tuberculosis is not found in the sputum. He points out that the basis of the changes produced by the gases is a proliferative fibrosis which destroys the protective powers of the bronchi, and leads to an invasion by organisms, and while tuberculosis may play a part in the invasion, he considers it a very minor one. It is probable that the case, described earlier in this paper, in which tuberculosis was suspected was really one of fibrosis such as Hawes has observed.

4. In nearly half the cases (45 per cent) there is definite tachycardia, and the question of the causation of this condition, upon which prognosis and treatment depend, appears to me to be the most important problem in connection with the subject. In 85 per cent of these tachycardia cases no sign of anything abnormal was found on examination of the heart. Is there any definite myocardial change, or is it due to some other cause? Captain R. A. Peters, R.A.-M.C., in a report of the Chemical Warfare Committee, states that the changes in the heart in recent cases are secondary to the changes in the lungs, and are due to (*a*) increased resistance in lungs, and (*b*) oxygen want; but it is difficult to understand how this can apply to cases of some years' standing, in many of

[1] British Journal of Surgery, 1915–1916, iii, 234.

[2] Boston Medical and Surgical Journal, 1921.

which there are no symptoms of any lung trouble. Tachycardia is undoubtedly more marked in cases of poisoning by the phosgene group. It may be functional only, or it may be due to a persistence of the polycythemia which is found in recent cases.

5. The persistence of the gastric symptoms also raises the question of organic or functional causation. The evidence of any actual gastritis or chronic catarrh is not clear, and the nausea and vomiting may be merely functional.

6. Why should neurasthenic symptoms supervene on gas poisoning? Undoubtedly a certain number of cases do develop neurasthenia in a mild degree, but it is not clear whether gas poisoning has a direct causal relationship, or whether the neurasthenia is due simply to the shock to the nervous system at the time of the original casualty.

7. Another interesting point is the pain in the chest of which complaint is so frequently made. Is such pain any evidence of myocardial involvement, or is this also merely a functional manifestation?

8. Lastly, with reference to prognosis, more especially in those cases in which physical signs are absent or very indefinite, does the element of "pension" operate as a factor in the causation or continuance of symptoms?

These figures give a very good idea of the amount of invalidism as compared with the total number of gassed cases, as well as the condition of these patients four or five years after they were gassed. They indicate that at the worst, not more than 12 per cent of all gas casualties suffer from any permanent disability. I say at the worst because these figures include the casualties produced by the early cloud attacks of chlorine when the victims had no protection whatever, and also the large number of casualties produced by mustard when this gas was first introduced, was not understood, and consequently when most of the troops exposed to it had either no protection or totally inadequate protection. Obviously when efficient protection is afforded, the percentage of casualties that will be so severely affected as to suffer permanent disability, will be very greatly reduced.

We may now examine the figures indicating the amount of disability produced by the different gases.

Chlorine. Meakins and Priestly analyzed 700 consecutive cases of chlorine poisoning of patients gassed in the 1st Canadian Division north of Ypres during April and May, 1915. Of these 700 cases, 368 either died in France or were returned to duty. The remaining 332 were transferred to England. The ultimate disposition of these cases was as follows:

Returned to France for duty	80 or 24.1 per cent
Retained in United Kingdom for duty	48 or 14.5 per cent
Invalided to Canada	204 or 61.4 per cent

Of the 204 invalided to Canada, 16 were invalided for causes other than gas poisoning. The remaining 188 cases were investigated four years after gassing and their records showed the following disabilities:

No appreciable ailment	54
Irritable heart	78
Bronchitis and asthma	26
Neuroses	18
Not traced	12
	188

From these figures it will be seen that of the total 700 cases, 122 or 17.4 per cent suffered some permanent disability, and that of these disabilities approximately 11 per cent were caused by irritable heart, while between 3 and 4 per cent were caused by bronchitis and asthma. For reasons given above these figures probably present the highest possible percentage of disability that can result from gas poisoning.

On the other hand analysis of mustard gas casualties by the same investigators showed a remarkably low percentage of permanent disabilities. Meakins and Priestly analyzed 4575 cases of mustard poisoning transferred to hospitals of the United Kingdom. Of these, only 787 or 17.2 per cent of the total remained in hospitals longer than nine weeks. There were no deaths after the eighth week. The symptoms responsible for hospitalization after the ninth week were:

Photophobia, functional	131
Bronchitis	118
Laryngitis	107
Irritable heart (functional?)	116
Burns	39
Gastritis	35
Conjunctivitis and bronchitis	90
Unknown	75

Only 76 cases remained in hospital more than four months, and only 0.5 per cent of this group, or 0.08 per cent of the total 4575 cases were discharged as permanently unfit. In this small percentage of cases the causes of disability were bronchitis, irritable heart and neurasthenia. It is possible that this small percentage of cases might be permanently partially disabled, though of this there is no proof whatever, other than the observation that few cases ever recover after they have once been admitted to a pensionable status.

Wilson and Mackintosh (Quarterly Journal of Medicine, 1919–1920, XIII, 201) analyzed a series of 1500 consecutive mustard gas casualties admitted to No. 7 Stationary Hospital between January 1 and October 31, 1918. Eighty-seven per cent of all mustard casualties were able to march half a mile with equipment within a month after gassing. Thirteen per cent of all mustard casualties were sent to England. In these cases the period of invalidism lasted from three to six months.

Early in 1919 many months after the men had been sent to England as the result of gassing, they were examined. Those who had not fully recovered fell into three groups:

1. Men with functional disorders and without organic lesions.
2. Men under treatment for effort syndrome.
3. Men with signs and symptoms of lesion in the respiratory tract.

It is obvious that men in the first group could be restored to health. With regard to group two, the authors state that of the entire 1500 cases, 125 men at first complained of effort syndrome, or disordered heart action. All but 19 of these men were relieved by a short treatment of graduated exercise. In 14 of these men the symptoms were due to other conditions or had existed before the war, and only 5 were directly due to gas.

In the third group there were a few cases of emphysema, some of which may have existed previous to gassing. A few cases were suspected of tuberculosis because of cough, muco-purulent sputum, slight hemoptysis and long continued evening pyrexia. But repeated x-ray examination and negative sputum led to a favorable prognosis. Practically all of these men recovered within nine months. "Phthisis is no more than a curiosity as a complication after gassing. We have seen but one sample in our series."

A number of articles have appeared in French literature concerning the sequels of gas poisoning. One of the best of these is by Achard (Bull. de l'Acad. de Med., 1919, LXXXI, 135).

Between February, 1917, and December, 1918, he observed 3535 cases as follows:

Vesicant gas	2958 or 84 per cent
Suffocant gas	567 or 16 per cent

Although cases of vesicant gas are more frequent, the sequels are more frequent and serious in suffocant gas poisoning. A considerable

number of these latter cases suffer from purely functional disturbances such as cough, substernal pain, limited respiration and nocturnal dyspnea. In another group of cases these functional disturbances are more severe and in addition physical signs are present. There are two general types. (1) The emphysematous; the thorax is globular and sonorous. Inspiration is feeble and expiration is prolonged; râles are heard all over the chest. (2) The bronchitic or pseudotuberculous. There is cough, expectoration of muco-purulent sputum and sometimes slight hemoptysis. The general health is poor and there may be a loss of from 8 to 15 kilos body weight. Some patients have an evening rise of temperature that may last for months. But careful and repeated examinations show that there is no tubercular infection. On x-ray examination the lungs are clear which is a sign of great diagnostic importance especially when the apices are clear. In the hilum there are no shadows of the adenopathy so characteristic of tuberculosis. The sputum contains no albumen and tubercle bacilli are never found. Finally the course of the disease settles the question. After a long time which in some cases may be months, a year or even more, the fever falls and the physical signs disappear, and most of such cases eventually make a complete recovery.

With regard to vesicant gas, similar cases are found but generally recover sooner. Few cases remain after nine months.

Personally Achard saw only 6 out of 3535 cases where tuberculosis developed after gassing, and says that the number of cases developing active tuberculosis after gassing is quite small as compared with the number of cases among the line of the army that were not gassed.

Experimentation which he performed on this point was in accord with the clinical facts just stated. Two lots of 20 guinea-pigs were inoculated at the same time with the same dose of B. tuberculosis. One lot served as a control, and the other lot was gassed with a non-lethal dose of phosgene (0.5 cc. per cubic meter) for from five to ten minutes every eight days during five months. At the end of nine months the control lot had lost 7 animals and the remaining 13 were killed. The lesions of tuberculosis were marked in all 20. Among the gassed animals only 5 had died and the remaining 15 were killed. In these animals the lungs presented the signs of subacute pulmonary edema, but the tubercles were neither more or less abundant than in the lungs of the control animals.

Digestive disturbances. These may persist in chronic form for some time after gassing. Anorexia, intolerance for certain foods, hyperchlorhydria, nausea, meteorism, and constipation or diarrhea are all frequent symptoms. But they rarely last more than one year after suffocant gas. Achard observed only one case eighteen months and one case twenty-four months after gassing. The duration of these digestive disturbances after mustard gassing is even shorter. Almost all such cases recovered completely in six months. In only 6 cases did the condition persist longer than ten months.

Cardiac disturbances. In a very few cases, disordered heart action may persist for a long time, which is about the same for the suffocant and vesicant gases. Eight such cases out of a total of 567 have been observed following suffocant gases: 5 were observed between one and two years after gassing, and 3 between two and three years after gassing.

Seven such cases out of a total of 2958 were observed following vesicant gas. Three cases were observed ten months after gassing, and 4 in the course of the second year.

These figures are in essential harmony with the figures previously quoted from British sources.

We also possess similar studies made on American gassed cases.

Soon after the Armistice, a Board of medical officers was convened at Fort Grant, Illinois, to examine all returning soldiers claiming disabilities from the effects of gas. Robert S. Berghoff, review examiner of this Board published his observations ("The more common gases; their effect on the respiratory tract." Archives of Internal Medicine, 1919, 24, p. 678.) The history of each case was carefully studied, followed by a careful physical examination. As a result of such observation of 2000 cases, Berghoff summarized his findings and conclusions in the following classifications:

1. The apparently normal group which included about 50 per cent of the total. In this group a careful, thorough physical examination failed to reveal any abnormalities; expansion of the chest was normal; breath sounds clear and vesicular; percussion note negative; no moist sounds, but, in the face of these apparently normal physical findings, the men complained of cough, shortness of breath on exertion, etc.

2. The second group was the bronchitic group which included about 30 per cent of cases examined. The findings in these cases were rather definite and did not differ materially from the subacute

or chronic bronchitis early encountered. The type of breathing was harsh and high-pitched throughout. Expiration was prolonged and at times interrupted, but most pronounced after exercise. Moisture in these cases was abundant. There were coarse moist râles rather evenly distributed at the bases of the lungs.

3. The third group was characterized by the presence of emphysema. On inspection, the chest seemed moderately rigid, the movements of the diaphragm limited; expansion impaired and accessory muscles thrown into use. On palpation tactile fremitus was markedly diminished and in some instances entirely absent. On percussion a hyperresonant sound was detected. On auscultation the breath sounds over the entire chest except the bronchial areas were much diminished. In some cases discrete patches of emphysema could be mapped out.

After a general review of all cases by the Board, its findings were as follows:

1. That gas victims, irrespective of the type of gas and severity of attack sustained, showed no marked predisposition toward active pulmonary tuberculosis, or towards the re-activation of a healed or quiescent pulmonary lesion.

2. That gas victims presented little evidence of material destruction of lung tissue.

3. That gas victims with emphysema findings had a more protracted convalescence than those of the bronchial group.

On page 103 of the Surgeon General's Report for 1920, there is found the following paragraph:

> One hundred seventy-three cases of tuberculosis occurred during 1918 among the 70,552 men who had been gassed in action. Of this number, 78 had been gassed by gas, kind not specified; 8 by chlorine; 65 by mustard; and 22 by phosgene. The number of cases of tuberculosis for each 1000 men gassed was 2.45. Since the annual rate of occurrence for tuberculosis among enlisted men serving in Europe in 1918 was 3.50, and in 1919, 4.30 per 1000, it would seem to be apparent that tuberculosis did not occur any more frequently among the soldiers who had been gassed than among those who had not been.

It will be seen that the Surgeon General was very guarded in his final statement. Actually in the year 1918, there were $1\frac{1}{2}$ times as many cases of tuberculosis per 1000 among all troops in France as there were among those gassed; while in 1919 there were $1\frac{3}{4}$ times as many tuberculosis cases per thousand among all troops as occurred among the gassed cases.

Lt. Colonel H. L. Gilchrist, M.C., made a very extensive study of this whole subject, including an analysis of over one thousand applications for compensation filed with the Veterans' Bureau. This was published by the Chemical Warfare Service under the title, "Reports on the after effects of warfare gases." In this report Colonel Gilchrist concludes that those who apply for compensation a year after discharge claiming to have developed tuberculosis as a result of their army service certainly have no basis for such an assertion. They probably would have become tubercular patients even if they had never been in the army. If a year or so elapses from the time of the alleged gassing until symptoms of tuberculosis develop, it is quite probable that gas had nothing to do with the question provided that the soldier's lungs were clear at the time of discharge.

In conducting this investigation several facts have become apparent.

1. That neurasthenia has played a most important part; as a result, many men who were really gassed, although very slightly, sincerely believe that this factor is the cause of their illness.

2. That a large number of individuals were undoubtedly substandard in the beginning and would quite probably have broken down under any stress or strain.

3. That the generally unknown effect of warfare gases has furnished many individuals a peg upon which to hang all manner of symptoms.

4. That the period of time between our entry into the war and the present date, has offered ample opportunity for the onset of diseases totally unassociated with war wounds or exposure.

5. That a great many confuse the after effects of warfare gases with those resulting from the severe epidemic of respiratory diseases prevalent throughout the world during the gas warfare period.

The following testimony was given by Dr. Albert P. Francine before the Senate committee investigating the United States Veterans' Bureau, and was published in the Military Surgeon, April, 1924, vol. 54, p. 470. Dr. Francine was a specialist in pulmonary tuberculosis and during the war was assigned as a consultant to the gas service in the Fourth Army Corps.

Major General O'Ryan: Well, now, please give your views to the committee based upon your experience before the war and during the war with tuberculosis cases, and as a result of your observations of the effect of gas upon the human structure, the tissues, giving your views of the relation between gas poison and tuberculosis?

Dr. Francine: I have seen a large number of men who gave a history of gassing, and who attributed their symptoms to gas, and who claimed to have tuberculosis, who believed they had it, and who had been told they had it, and that diagnosis has been made very widely. I think that of such cases the error in diagnosis is over 50 per cent at least.

It has become increasingly apparent that such an error is not so likely to be made now, because these gas cases are clearing up; and we now have a definite picture of the symptomatology and physical signs of the effects of gas as distinct from the effects of tuberculosis. Though they are similar in certain respects, and so similar, in fact, that it is not at all surprising that a number of men, particularly men who did not study their cases carefully, and perhaps men of not very broad experience, should have made this error in diagnosis.

During the war, of course, I saw very intimately the picture of gas in the field and in the hospital. And this having interested me from the professional point of view, I made a special point of studying the lungs of these gassed men, both clinically and at post-mortem in the gas hospital. Clinically the acute picture of gassing, this mass inflammation, disguised the picture at times so that you could not clinically have diagnosed tuberculosis had it been present. But you could examine the sputum. Many of these men spat blood, and their sputum was uniformly negative.

But even more convincing than that, a number of cases, I may say a great many of these men died. Not relatively a great many, but absolutely. The percentage of death from gas was less than 2 per cent. And I went to the post-mortem room every day and examined the lungs. I was not so interested in the effects of gas elsewhere after I once knew it, but I examined the lungs to see whether there were latent tuberculosis lesions; that is, lesions that did not give symptoms, such as any of us here may have, and whether there was any evidence of these lesions, pathologically, having been lit up by this more or less intensive inflammation. I do not mean at the time of the gassing, but I mean following gassing, when one would expect it. While we were being rushed, and were too rushed to keep records, I suppose I can say that I saw then 100 to 125 posts. In a number of these cases it was perfectly and palpably evident to a man of pathological training that there were healed, nonactive lesions, which is not an uncommon occurrence, as you know. It is considered that probably 60 to 90 per cent of people have at one time or another had a tuberculous infection. But these palpable latent lesions showed no evidence of activity, or reactivation, from four to six to ten weeks after the acute inflammation which is supposed to give rise to tuberculosis.

I was only in the hospital six or eight weeks, but there were a number of men unevacuable on account of their condition; they had been severely gassed, and were recuperating, were feeble, were skin and bone, and so on, and they died from an intercurrent infection, which was rampant, as you know, influenza and broncho-pneumonia; and they died, not in any sense due, except indirectly, to the gassing, but to this cross-infection. So that those men would have had plenty of time to develop signs of activity in those latent tuberculous lesions. For it takes anywhere from, we will say, two to three weeks for such evidence to appear macroscopically, grossly; for tubercles and localized inflammation to appear, indicating activity; and such would have developed

by the time I saw them, if gas had had any effect, any effect whatever, in developing already existing tuberculosis or activity in a latent or healed state.

There was a similar group of cases who were orderlies. We were very short of nurses, and I did not discharge a certain group of men who had been gassed, but whom we had to keep in the hospital to help out as orderlies; these were quite a group. These men had been gassed, but were able to be about and help, carry trays, and so on. Some of these men died through infection, quite a number of them. And these men, again, showing milder pathological evidence of gas, having a history of gas, showed no sign of reactivation in latent lesions. And in the cases I examined post-mortem, I should say there were at least 20 to 25 men who showed latent or healed lesions—that is, perfectly plain latent lesions—and in not a single case was there any evidence of activity developed through gas.

I believe that so far from producing tuberculosis—anybody might, indeed, think so on account of the local irritation in the lungs and bronchi—gas may tend to prevent tuberculosis, and I will tell you why. Because its primary action is to produce congestion of the lungs. In fatal cases it causes severe inflammation followed by congestion and edema and prevents the aeration of the blood, and quickly smothers so to speak, the patient. He cannot get air into his lungs or blood. When the man drowns or smothers, he is suffering primarily from that inflammation caused not by a germ but a chemical. We know that a nonbacterial congestion or hyperemia is a favorable factor in tuberculosis. In other words, if we could produce locally in the lungs a congestion, we could go far toward curing tuberculosis. People with heart disease (I speak of this simply to you gentlemen as laymen, to explain some of my reasons)—people with heart disease, that type of heart disease known as mitral stenosis, where there is an increase of the blood in the lungs, do not suffer from tuberculosis. When I say that, I mean not generally; I mean it is exceptional. In my work in the hospitals in Philadelphia I saw many cases of mitral stenosis, and I do not remember one having tuberculosis. Some were thought to have it, but at post-mortem they did not, probably because of this congestion of the lungs.

For that reason I feel justified, at least, to express my opinion, earnestly arrived at, that if it has any effect, it may well be a tendency to prevent tuberculosis.

Further, it has been claimed clinically that men who have tuberculosis and have been gassed run a more favorable, slower, more indolent course, are better off over the same period of time, improve more, than the same type of cases having tuberculosis but who have not been gassed.

THE CHAIRMAN: You mean that there are not a great many cases of permanent injury to the lung tissue from the inhalation of gas?

DR. FRANCINE: I do mean just that.

THE CHAIRMAN: Are there no cases, Doctor?

DR. FRANCINE: The permanent effect of gas is the result of this irritation, this congestion; and what follows all inflammations and congestions, if they

last long enough, is, broadly speaking, the destruction of the finer cellular tissue in places and its replacement by fibroid tissue.

And to that extent these post-influenzal and post-gas lungs show, in the very bad cases, the remains of this thing, and it is still being mistaken for tuberculosis very widely. But they clear up. You take a man that has been gassed and x-ray him when he has recovered from acute symptoms. A plate taken two years later shows, instead of these dark shadows, marked evidence of clearing up. And they do clear up. Their health and strength improve markedly, too.

As a matter of fact, the permanent effect of gas is, I believe, more serious on the morale and on the heart. These men are astounded and shocked by this unknown thing. They may be very sick for a while. Later they suffer with what you would call a nervous heart, an irritable heart—what we call an N.C.A. These symptoms improve slowly. But they suffer with palpitation on exertion, rapid pulse, shortness of breath, faintness, nervousness, disturbed sleep. That is what they suffer with. And then as time goes on they get better, though they may have a chronic cough. But the damage done to the lungs is not the thing that produces, as a rule, very trying symptoms. In other words, you can perhaps better understand that, when I say that if you could remove one lobe of your lungs you would not suffer a great deal from loss of air space. There is a great deal of tissue there that you don't need, so to speak, and the amount of fibroid tissue that is developed in some of these gassed lungs, around the bronchi and beyond, is not of itself very handicapping. Not the mere fibroid tissue alone.

The Chairman: To boil it all down, it comes to this, does it not, that the cases of tuberculosis which have developed within the last two or three years among the veterans of the World War, who were gassed, ought not fairly to be ascribed to the gassing as the primary predisposing cause. Is that your conclusion?

Dr. Francine: Yes; that is my conclusion, of course. It is the conclusion of every man who knows.

In concluding the consideration of these reports concerning the ultimate condition of men who were gassed, it seems quite apparent:

1. That only a very minute percentage have suffered from any permanent disability directly caused by gas.

2. There is not the slightest evidence that gassing has increased the liability to tuberculosis. So far as the evidence goes, men who have been gassed suffer less from tuberculosis than normal individuals under the same circumstances.

3. That neurasthenic conditions are responsible for a considerable part of the disability alleged to result from gassing.

4. That few of the cases that receive a pension will ever admit a subsequent improvement or cure.

OBSERVATIONS UPON THE ACTUAL RATE OF RECOVERY IN EXPERIMENTAL ANIMALS

These observations have been made by the Medical Research Division at Edgewood Arsenal.

Method. The dogs used received about the lethal concentration of the gas in question for the period of time exposed, and a large number of these dogs died acutely from the effects of the gas. Those that survived were obviously sick for periods of time varying from a few days to a month. They were considered to be recovered after their temperature, pulse and respiration had become normal, and after all symptoms had disappeared. Such dogs were then kept in kennels with an exercise yard under conditions as nearly normal as possible, and killed at varying intervals after recovery. They were then carefully studied to determine what pathological changes remained as the result of the gassing. It will be seen at once that this is a very severe test. In the first place since the concentrations employed were in general lethal, and the majority of dogs used died acutely, it is obvious that the survivors had been exposed to far higher concentrations of gas than were the great majority of human cases that were gassed in the field. In the second place, the criterion of pathological changes is extremely severe. For it is obvious that complete functional recovery might follow in many cases in which changes could still be demonstrated microscopically. As Dr. Francine pointed out we could probably lose an entire lobe of the lungs without missing it.

The animals were killed by injecting 10 cc. of saturated solution of magnesium sulphate into the left ventricle. This causes instantaneous stoppage of the heart, immediate death without any agonal convulsions or other circumstances that would produce changes in the lungs.

The lungs of dogs are rather delicate and cannot be much handled without producing artefacts. Therefore when the body was opened, the trachea, and both lungs were removed, and Zenker's fluid was poured into the trachea until the lungs were normally expanded, at the same time immersing the lungs in a vessel containing Zenker's fluid. By this method fixation is complete and almost instantaneous. Sections were cut through blocks taken from various portions of the lungs after the usual technique of embedding in paraffine, stained in general with hematoxylon and eosin and studied.

RESULTS OBTAINED BY KILLING DOGS AT INTERVALS

From this table it may be seen that:

Phosgene recoveries

NUMBER	LENGTH OF TIME RECOVERED	FINDINGS
1	2 weeks	Fibrino-purulent pleurisy, purulent bronchitis
2	2 weeks	Bronchi plugged, atelectasis
3	2 weeks	Atelectasis
4	2 weeks	Hemorrhage, plugging of bronchi, atelectasis
5	2 weeks	Pneumonia
6	2 weeks	Extensive edema, hemorrhage, plugging of bronchi, atelectasis
7	2 weeks	Extensive hemorrhage, plugging of bronchi, atelectasis
8	3 weeks	Obliterative bronchiolitis, atelectasis
9	3 weeks	Edema, hemorrhage, atelectasis
10	3 weeks	Plugging of bronchi, atelectasis
11	3 weeks	Bronchi plugged, atelectasis, obliterative bronchiolitis
12	3 weeks	Congestion, plugging of bronchi and atelectasis, all mild
13	3 weeks	Hemorrhage and edema
14	3 weeks	Congestion, hemorrhage, edema, plugging of bronchi, atelectasis
15	3 weeks	Congestion, hemorrhage, plugging of bronchi, atelectasis
16	4 weeks	Recovered
17	4 weeks	Old scars, plugging of bronchi, and atelectasis
18	4 weeks	Some plugging of bronchi, and atelectasis
19	4 weeks	Atelectasis
20	4 weeks	Edema, patches of organization, desquamation of alveolar epithelium
21	4 weeks	Nearly recovered. One patch of atelectasis with slightly plugged bronchus
22	5 weeks	Some plugging of bronchi, and atelectasis
23	5 weeks	Pneumonia
24	5 weeks	Plugging of bronchi, atelectasis, small patches of organization
25	5 weeks	Some plugging of bronchi and atelectasis
26	5 weeks	Pneumonia
27	6 weeks	Plugging of bronchi and atelectasis
28	6 weeks	Congestion and atelectasis
29	6 weeks	Plugging of bronchi and atelectasis
30	6 weeks	Plugging of bronchi and atelectasis
31	6 weeks	Recovered
32	6 weeks	Obliterative bronchiolitis, plugging of bronchi, slight atelectasis
33	6 weeks	Plugging of bronchi and atelectasis
34	7 weeks	Recovered. Fibrosis about bronchioles
35	7 weeks	Some plugging of bronchi with atelectasis

Phosgene recoveries—Continued

NUMBER	LENGTH OF TIME RECOVERED	FINDINGS
36	7 weeks	Hemorrhage, some plugging of bronchi, and atelectasis
37	7 weeks	Chronic bronchitis with plugging of bronchi and atelectasis
38	7 weeks	Slight plugging of bronchi and atelectasis
39	8 weeks	Recovered
40	8 weeks	Nearly recovered. One spot of organizing bronchiolitis
41	8 weeks	Nearly recovered. Slight plugging of bronchi and atelectasis
42	8 weeks	Plugging of bronchi, atelectasis, metastatic carcinoma from thyroid
43	8 weeks	Plugging of bronchi and atelectasis
44	8 weeks	Plugging of bronchi and atelectasis
45	8 weeks	Plugging of bronchi and atelectasis
46	8 weeks	Hemorrhage and edema
47	8 weeks	Edema
48	8 weeks	Mild chronic peribronchitis, atelectasis, slight edema
49	8 weeks	Pneumonia
50	9 weeks	Recovered
51	10 weeks	Recovered, minute patches of organization
52	10 weeks	Plugging of bronchi and atelectasis
53	11 weeks	Edema. One patch of pneumonia
54	12 weeks	Recovered
55	13 weeks	Congestion and atelectasis
56	16 weeks	Recovered
57	16 weeks	Obliterative bronchiolitis
58	18 weeks	Patches of organization
59	5 months	Recovered
60	5 months	Recovered
61	6 months	Recovered
62	6 months	One very small patch of pneumonia
63	8 months	Bronchi plugged, obliterative bronchiolitis, marked atelectasis
64	1 year	Recovered
65	15 months	Recovered

From this table it may be seen that:

1. A few cases show complete recovery: i.e., there are no pathological findings even after lethal concentrations in some cases as early as four weeks after gassing.

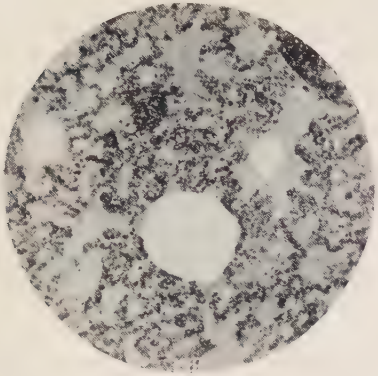
2. Five months after gassing, no pathological lesions can be detected in the majority of the animals studied.

3. No lesions could be detected in the two dogs killed one year and 15 months after gassing.

4. Although this series is by no means complete, it is reasonable to conclude that very few individuals, have any pathological basis for symptoms more than a year after even the most severe gassing with phosgene.

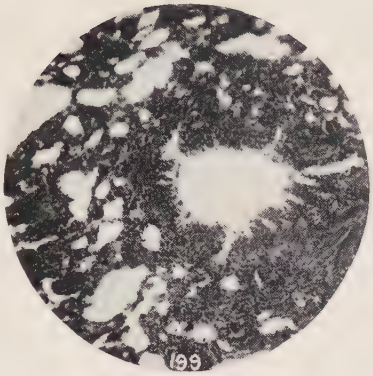
Mustard recoveries

NUMBER	LENGTH OF TIME RECOVERED	FINDINGS
1	Killed at time of recovery	Recovered
2	10 days	Edema, atelectasis, subacute bronchitis
3	2 weeks	Congestion, hemorrhage and edema
4	2 weeks	Edema, bronchi plugged with cellular debris, atelectasis
5	3 weeks	Edema, hemorrhage
6	4 weeks	Slight edema with plugging of bronchi and atelectasis
7	5½ weeks	Recovered
8	6 weeks	Nearly recovered. One spot showed plugging of bronchi and atelectasis
9	6 weeks	Edema
10	7 weeks	Edema and hemorrhage
11	8 weeks	Spots of plugged bronchi and atelectasis
12	8 weeks	Some plugging of bronchi, atelectasis. Otherwise normal
13	9 weeks	Recovered
14	9 weeks	Recovered
15	9 weeks	Recovered. Slight chronic infiltration of bronchial walls
16	10 weeks	Recovered
17	10 weeks	Recovered
18	11 weeks	Congestion, hemorrhage, edema
19	13 weeks	Recovered
20	13 weeks	Recovered
21	13 weeks	Old scars, some atelectasis
22	14 weeks	Recovered
23	16 weeks	Recovered
24	16 weeks	Bronchi plugged
25	16 weeks	Plugging of bronchi and atelectasis
26	16 weeks	Some plugging of bronchi and atelectasis
27	20 weeks	Spots of edema
28	22 weeks	Three out of six sections show edema. Otherwise normal.
29	22 weeks	Edema
30	29 weeks	Recovered
31	35 weeks	Recovered
32	9 months	Large scars. Several minute patches of "chemical pneumonia"



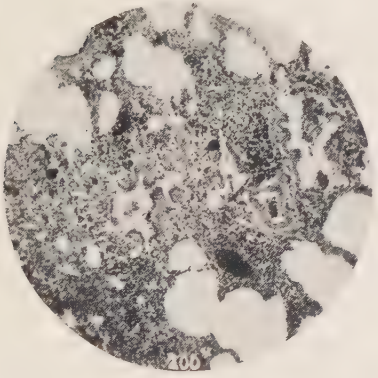
PHOSGENE

Death in 24 hours. Edema and congestion.



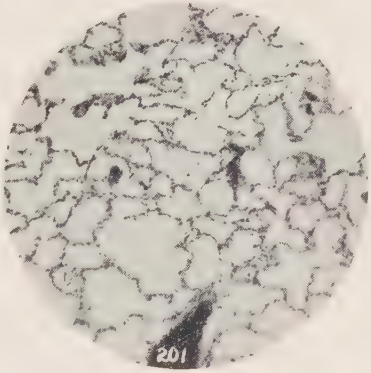
PHOSGENE

Killed after 3 weeks. Partial atelectasis.



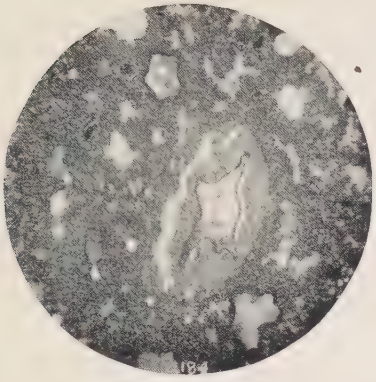
PHOSGENE

Killed after 11 weeks. Partial atelectasis, chronic bronchitis.



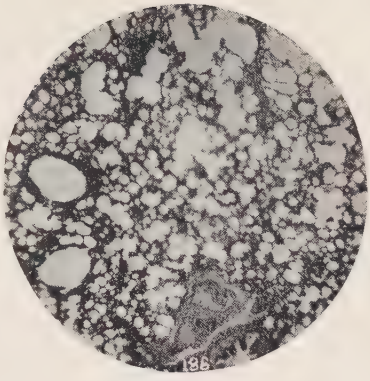
PHOSGENE

Killed after 20 weeks. Recovery



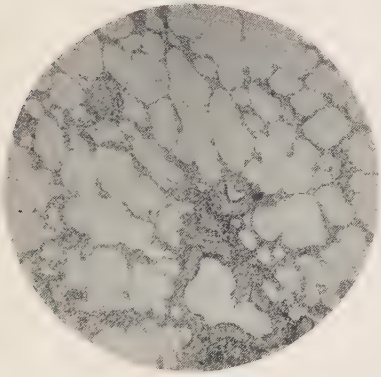
CHLORPICRIN

Died in 9 hours. Congestion, hemorrhage, edema.



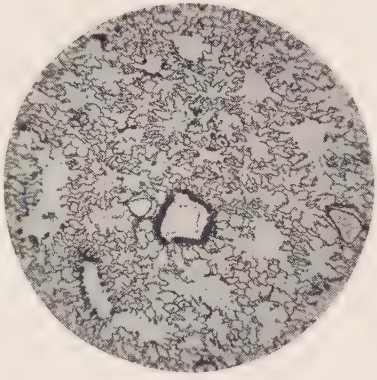
CHLORPICRIN

Killed after 2 weeks. Partial atelectasis.



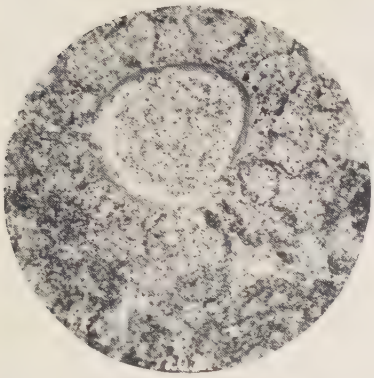
CHLORPICRIN

Killed after 26 weeks. Organization and thickening of alveolar walls.



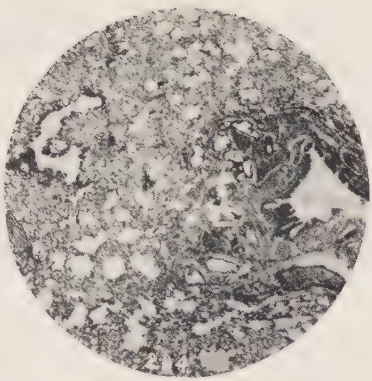
CHLORPICRIN

Killed after 4 weeks. Return to normal.



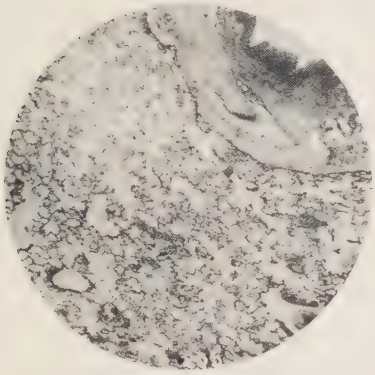
MUSTARD

Death in 8 days. Purulent bronchitis, broncho-pneumonia, congestion, hemorrhage.



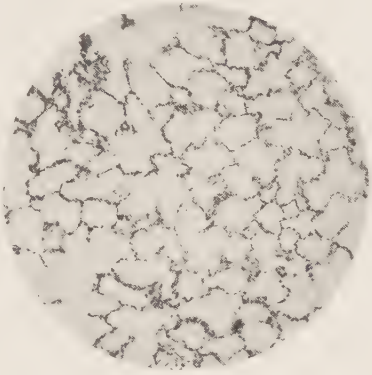
MUSTARD

Killed after 7 weeks. Partial atelectasis, hypertrophy of bronchial epithelium, dilatation of mucous glands.



MUSTARD

Killed after 10 weeks. Hypertrophy of bronchial epithelium, dilatation of mucous glands, normal alveoli.



MUSTARD

Killed after 16 weeks. Recovery

From this table which only includes dogs observed up to nine months, it may be seen:

1. That there is no point up to nine months at which it can be said that the majority of the dogs show no pathological lesions. Recovery from lethal concentrations of mustard is evidently a longer process than recovery from lethal concentrations of phosgene.

2. However, even after such lethal concentrations of mustard, in 13 out of 32 dogs, or 40 per cent, no lesions could be detected as the result of gassing.

3. Bearing in mind the fact that very few men have ever been gassed with such lethal concentrations in the field, it seems reasonable to conclude that only a very small percentage of men will present any pathological findings more than eighteen months after gassing by mustard.

A similar study of 59 dogs gassed by lethal concentrations of lewisite, has shown that complete recoveries begin to occur as early as six weeks after gassing, and that the majority of the dogs present absolutely no lesions twelve weeks after gasing. This table is not given *in extenso* because lewisite was not used during the war, and consequently there are no human cases in which this gas can be responsible for possible disability.

In conclusion, it may be said that the findings on these animals corroborate the opinions expressed by the observers who have recorded their study of human cases. While these animals were in the kennels they were very active, and with few exceptions apparently healthy, and suffered no respiratory embarrassment when they ran about. It was originally intended to study the effect of exercise upon the lungs and heart of these recovered animals, and a treadmill was constructed for this purpose. But the dogs were too intelligent to use it, and either jumped out or refused to run, and this part of the program was abandoned. But even the animals that had marked lesions when killed, with few exceptions suffered no distress while living. In the vast majority of human cases who are not gassed so severely, and who receive infinitely better care in hospital, all lesions must have disappeared a year after gassing, and such minor lesions as may remain in a minority of cases, can hardly be a cause of serious disability. By far the greater proportion of cases that suffer disability more than a year after gassing, are suffering from neurasthenic conditions, particularly functional disordered heart action, or a neurasthenic condition of the respiratory center. Treatment of such cases should be directed along these lines.

CHAPTER XVI

Naval Medical Aspects of Chemical Warfare[1]

The initial use of poison gas in warfare is shrouded by the mists of time. It was probably first used by primitive tribes in smoking each other out of caves in which they had taken refuge. That it was regularly used in very remote periods is shown by Assyrian bas-reliefs in the British Museum which depict the use of liquid fire, and it was often used in warfare both by land and sea throughout the ensuing ages. Its use is described at the siege of Syracuse, 413 B.C. (1), and at the siege of Rhodes, 304 B.C. (2). Throughout ancient times agents similar to the above were used, partly for their asphyxiating effect, and partly as incendiaries. They appear to have been composed of various mixtures of pitch, sulphur, petroleum and lighter oils and do not seem to have been particularly effective.

But with the introduction of "Greek fire," chemical weapons assumed a more important position in war, and especially in naval warfare.

About 668 A.D. an architect named Kallinikos fled from Heliopolis to Constantinople and imparted the secret of "sea-fire" to the rulers of the Eastern Roman Empire. With the aid of this new agent the Greeks defeated the Arabs repeatedly and its formula was jealously guarded for over five hundred years.

Many formulas have been given for "Greek fire." The one which is now most generally accepted is that given by Groot (3), who states that it consisted of sulphur, naphtha, wax, oil of balm, and quicklime. As quicklime develops a temperature of 150°C. on contact with water its heat of reaction would be sufficient to ignite the light oils used, and history graphically describes the asphyxiating odors and the series of small explosions occurring during a Greek fire attack. The liquid fire was projected from a tube or siphon mounted on a swivel in the bows of the Byzantine vessels, being forced through the tube by a large hand pump similar to the present fire-engine hand pump (4). Liquid fire was never used successfully by anyone but

[1] This chapter has been written by Lieut. Commander Duncan C. Walton, (M.C.), U. S. N.

the Byzantine emperors, and attempts by others to utilize it have uniformly failed. Among such failures may be mentioned the attempt of the Union army before Charleston during the Civil War (5), and the ineffective "Flammenwerfers" of the Germans during the World War.

During the siege of Sevastopol by the allied armies in 1863 Admiral Dundonald suggested the burning of 400 or 500 tons of sulphur before the city, hoping to asphyxiate its defenders with the fumes of sulphur dioxide. The British government refused to adopt this means of warfare because of its inhumanity.

A somewhat similar scheme for overcoming the enemy has been in use for centuries among the pirates of the Eastern seas, who utilized "stink-pots" or "stink-balls," consisting of various containers filled with a mixture of pitch, resin, gunpowder, colophony, asafetida and other irritant chemicals. These "stink-pots" produced a marked degree of disablement from coughing and lachrymation when ignited and thrown on the crowded decks of enemy vessels.

Matthew Paris in "Historia Anglorum" states that during the reign of Henry III, the English commander, in an engagement with a French fleet, maneuvered his ships to windward, sailed down wind, threw a large amount of finely powdered quicklime on the French vessels and by this "strategem" won the day.

Beebe (7) has collated from history several other quaint forms of chemical and biological attacks, including such curious methods as the hurling of earthen pots filled with poisonous reptiles on the enemy decks.

From the preceding brief sketch it can be seen that while Chemical Warfare was of importance in ancient naval warfare it has not been used in modern naval engagements. But during the World War the personnel of all the navies engaged were equipped with gas masks to be prepared against a surprise attack and on several occasions these masks proved to be of great value. During the attack of H. M. S. *Vindictive* on the Mole at Zeebrugge, April 22 and 23, 1918, the crew were subjected to gas, and McCutcheon (6) makes the following statement: "Gas masks—these were found to be useful in every part of the ship, but especially so between decks. While the ship was alongside the Mole, unpleasant effects were experienced from some gas, the nature or origin of which we were unable to determine. The chief symptoms were smarting of the eyes and coughing." Of the Germans, Schwarte (8) states "In the battle

of the Skagerrack the fleet used with good results the 11/11(box) mask to protect against the effects of nitrous and organic nitrous bodies."

That chemical warfare has never been used at sea in modern times for other than screening purposes is a source of surprise to many. The explanation is simple. Following the re-introduction of chemical attacks in 1915 by the Germans, the Navies of both contending forces, while preparing themselves against a surprise attack by the enemy did not wish to be responsible for the initiation of poison gas warfare at sea. This was from very much the same reasons that none of the contending air-services ever used air-craft gas bombs against the unprotected populations of enemy cities, i.e., partly from a sense of chivalry but mainly from a wholesome fear of retaliations.

In addition to this both sides felt grave doubts of the value of the new agents as compared to the older methods. The results of a direct hit by a shell, a torpedo or a bomb loaded with high explosive were known. The results to be expected from the use of chemical agents at sea were problematical and the abandonment of arms of known value in favor of new and untried methods was not considered justified.

But with the return of peace in 1918 naval strategists in considering the lessons of the World War were compelled to envisage the possible use of poisonous chemical agents by an enemy force in the future. A surprise attack of any nature is always of the greatest value, and a surprise attack with chemical agents, skilfully delivered and resolutely followed up might well deliver the control of the seas to the attacker. This would be especially the case if some new chemical were employed, or if any of the old agents were laid down in some newer and more efficient manner. That the danger is very great none can deny and a study of certain army tactics in the presence of gas lends credence to this view. For example, any army force would immediately evacuate an area drenched with a persistent vesicant, being secure in the knowledge that the enemy could not enter it either. Needless to say such tactics cannot be employed on a vessel, which leads to the necessity for a high degree of protection for naval vessels. For if not so protected it would be but a short time before every one of the personnel so closely herded aboard would become a casualty. Furthermore, the army by using gas proof dugouts can protect their forces to a certain extent, while the present ventilating systems of naval vessels would merely serve to thoroughly distribute poisonous agents through all compartments.

And it can readily be seen that while properly based armies can soon rush up supplies to strengthen their defense against new chemical agents this will often be impracticable in a fleet based on some foreign port. It follows from this that in time of war ships should be as thoroughly prepared in regard to anti-gas defense and supplies as possible, for there will be no time during a chemical attack to improvise defenses. In consequence the duty devolves upon medical officers to be prepared to diagnose and treat gas casualties rapidly in order that as many as possible of the combatant personnel be returned to their action stations immediately.

For medical officers on duty with marines ashore the Army chapters of this book provide instructions for the methods of handling gas casualties in the field. No attempt will be made in this chapter to outline the treatment of gassed cases, this being covered elsewhere in the book and only medical aspects of Chemical Warfare afloat that differ from Army problems will be considered here.

PROBABLE NAVAL USES OF GAS

The Naval medical officer should have a clear idea of what gases might be used against his ship, how they can be put over and when they would be used. Such knowledge will often aid him in making an early diagnosis of cases during a gas attack. The following outline covers probable Naval variations from the use of gas as employed by land forces.

1. Lung irritants

These will probably not be used. Most of them are non-persistent and if used prior to an enagement would not contaminate a ship for any length of time. Their action is slightly delayed so they would be of little value during an engagement, during which a direct hit from a shell or bomb filled with high explosive would be much more effective against personnel and material. But as it has been suggested that they might be used in shells of the secondary battery the medical officer should be prepared to handle cases gassed by lung-irritants.

2. Vesicants

A. Slowly acting vesicants such as mustard would probably not be used during an engagement but would be employed at other times to contaminate ships. The lung irritant and vesicant actions of this

class of compounds is so delayed that they would be of but slight value during a fleet action.

B. Immediate vesicants. No such compounds are known at present. If developed they could be used at any time.

3. Lachrymators

A. Non-persistent: As the non-persistent lachrymators are no more efficient, weight for weight, than the persistent lachrymators, they will probably not be used.

B. Persistent: These could be used at any time but would probably be used only during a naval engagement, to blind the enemy or at least compel him to mask.

4. Sensory irritants

A. Non-persistent: The same objections apply to non-persistent irritants as to other non-persistent agents. As the actual occupation or "boarding" of an enemy vessel seldom if ever occurs in modern naval warfare it will always be a persistent agent that is used.

B. Persistent: These can be used at any time, and especially during an engagement. Their action is quite rapid, irritant effects being experienced in from six to thirty minutes and the disablement produced by even very minute quantities is extreme. Even after settling out on the decks and bulkheads toxic smokes would later be raised as dust-clouds by the movements of the personnel and the presence of these persistent irritants in a compartment would render it uninhabitable.

5. Suffocants and paralysants

This group of gases, which includes such agents as hydrocyanic acid, cyanogen chloride, hydrogen sulphide, arsine and phosphine has been suggested as being of value in naval warfare as the closed spaces of a vessel would prevent their too rapid dissipation. Most of them are not practical for various reasons such as inflammability, difficulty of confining in shells and bombs or because they are too greatly decomposed by detonation. The possibility of their use and the possibility of the development of paralysant alkaloid derivatives should be borne in mind, however.

A general review of the above would indicate that practically all gases used at sea would be of a persistent nature and only those which act rapidly on the organism would be used during an engagement.

Smoke Screen by Destroyers. Irritant Smokes May Be Similarly Used

Hains (9), Fries and West (10), and Jones (11) have indicated thoroughly the ways in which toxic gases and smokes can be liberated at sea, and have suggested the following means:

1. Boxes and cylinders. *A.* To be used from aboard ship. Many types of these boxes were used during the World War for simple screening operations. They could easily be adapted to the release of toxic materials in clouds.

B. Floating smoke boxes. These were thrown overboard for screening purposes during the World War and were found to be of great value. If filled with toxic materials and released to windward of the enemy they should be effective.

C. Toxic smokes could be mixed with the ordinary screening smoke from the funnels as appears to have been done in the German attack on H. M. S. *Botha* (9).

2. Torpedoes. *A.* It is very doubtful if gas would ever be used in torpedoes fired for a direct hit.

B. The use of broaching torpedoes which would generate a cloud of toxic materials in the midst of the enemy fleet has been suggested.

3. Mines. The filling of marine mines with chemical agents offers little of value as an offensive weapon.

4. Shells. Probably none of the large calibre naval shells will ever be filled exclusively with chemical agents. But the use of a shell similar to the German blue cross shell which contained a small bottle of toxic smoke embedded in the high explosive filling, or the inclusion in shells of a small amount of lachrymatory substance would produce many gas casualties and compel the enemy to mask (12) in addition to the usual demolition effect.

5. Air-craft. Gases can be projected on vessels by any of the following means:

A. Sprays of persistent agents (11).

B. Curtains and screens of immediate irritants during an engagement (11).

C. By the dropping of float bombs to windward; such bombs can distil off a cloud of gas or smoke for from five to fifteen minutes (10).

D. Direct hits by bombs filled with persistent agents (10).

E. The sprinkling, with slowly hydrolizable compounds, of harbors or of places where landing forces are to be feared.

From a consideration of the above tabulations the medical officer should be able to judge fairly well to what class of compounds the personnel has been exposed. For example, during an engagement he

might expect lachrymators, toxic smokes, immediate vesicants and possibly suffocants and lung irritants, and could be reasonably sure that none of the more slowly acting chemicals would be used.

While the defense of the ship against chemical attack will be the duty of the anti-gas officer named for that purpose, certain medical problems will arise as a result of the defensive measures adopted. These problems can be considered under the headings of defense and of degassing.

INDIVIDUAL DEFENSE

1. Gas masks

A. Gas mask, Army type. Owing to the exigencies of ship life the personnel will not carry their individual gas masks at all times as the Army does. For purposes of cleanliness it will therefore become necessary to see either that the masks are tagged with the owner's name or that they are carefully disinfected before being stowed away.

As the side satchel containing the canister is inconvenient to the gun crews and others working in small spaces they will often sling the satchel on their backs, and personnel who are required to wear the bulky kapok coats will often simply abandon the use of the side satchel and place the canister in a pocket or slit in the coat. Great care should be exercised to see that these methods do not lead to kinks or constrictions in the corrugated tube with the resultant increased pressure drop and difficulty in breathing.

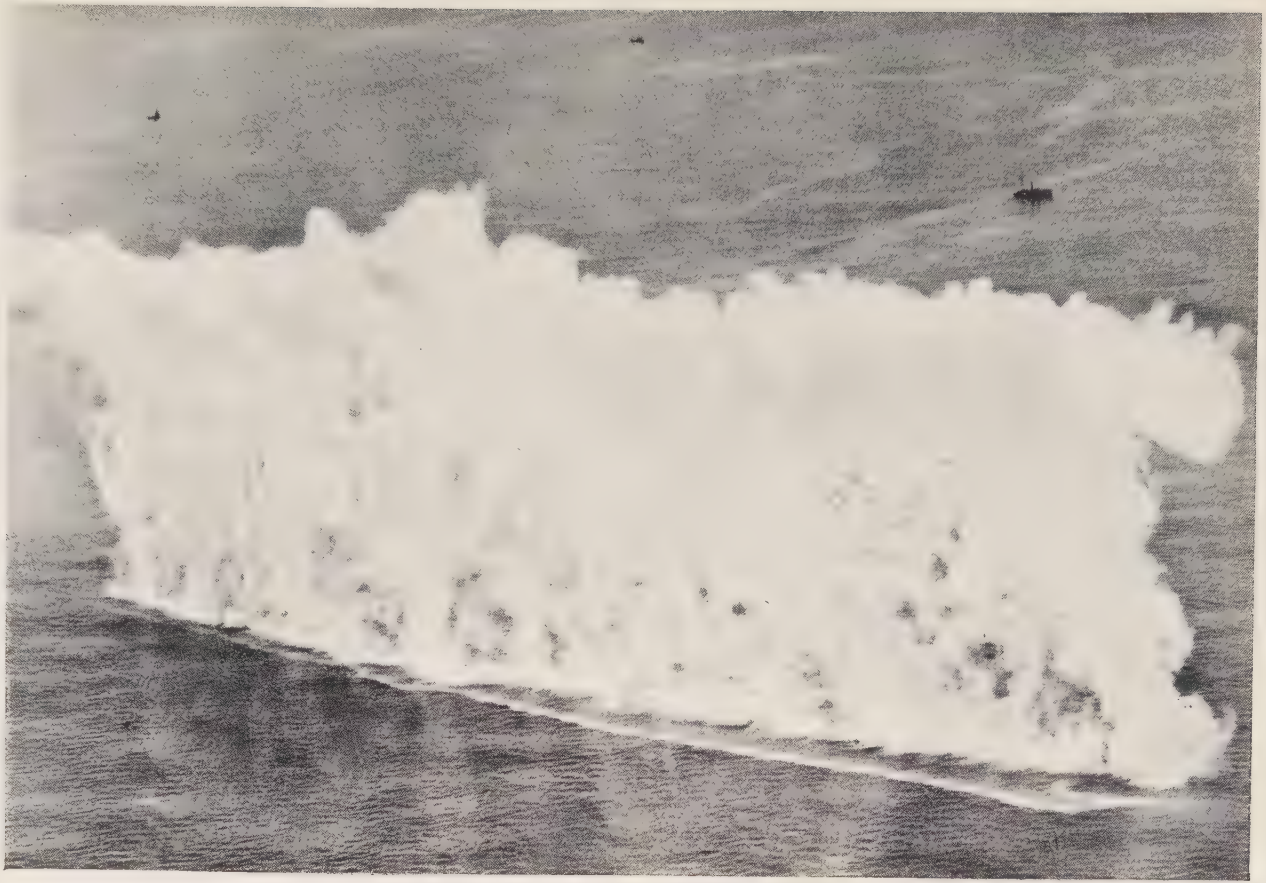
B. Diaphragm masks. These must be disinfected very cautiously to avoid weakening or perforating the diaphragm.

C. Optical masks. Masks used by the personnel manning the various optical instruments aboard should be carefully scrutinized to insure that the wearer's vision is as little interfered with as possible. Routine examinations of the visual acuity while wearing the mask are necessary to insure this and a change in the size of the mask or of its strap adjustment will often work wonders.

D. Extemporized masks. Should it become necessary to extemporize masks for a possible emergency the following method has been found of great value, the impregnating solutions used being those utilized by the French in their M_2 mask.

Twenty layers of muslin are soaked in a solution of

39 parts hexamethylenetetramine
37.5 parts glycerine



AIRPLANE SMOKE CURTAIN

27.5 parts nickel sulphate
11.8 sodium carbonate
Water sufficient to make a very thin mucilage

Twenty layers of cheese cloth are soaked in a solution of

107.0 parts castor oil
81.0 parts alcohol, 95 per cent
10.7 parts glycerine, 90 per cent
3.1 parts sodium hydroxide

Both sets of cloths are then wrung out till just moist. The layers are then placed over each other and the whole flat pad fastened over the mouth and nose by means of a strip of mosquito netting tied back of the head. This mask gives excellent protection against most war gases, including high concentrations of chlorine, phosgene or hydrocyanic acid.

2. Pastes and ointments

It may become necessary at times to prepare defensive ointments aboard ship to supplement deficiencies in the protective clothing. A formula for the best ointment as yet worked out can be found in Chapter VIII.

3. Clothing

Clothing impermeable to mustard can be extemporized from the oil cloth suits on board by sealing all openings with adhesive tape and fastening the garments tightly around the neck and ankles. Such clothing will give a fair degree of protection from mustard and similar vesicant gases and the gaps existing between the clothing and the gas mask face-piece can be covered with protective ointment. It should be remembered that after entry into a gassed compartment the clothing is contaminated and will have to be degassed. The use of rubber rain coats or boots is contraindicated by the fact that mustard is very soluble in rubber. Ordinary leather shoes and gloves can be made gas proof as follows. A mixture of

Carnauba wax.....30
Castor oil.....70

by weight is heated to 85° to 90°C. and the shoes and gloves put in for fifteen minutes; the excess is then drained off and the articles

dried in the drying room for two hours. Shoes and gloves so treated will protect against liquid mustard for two hours. After exposure to mustard these garments must be degassed with chloride of lime.

COLLECTIVE DEFENSE

From the view point of the gas defense officer a ship is considered as consisting of three kinds of stations for the personnel; closed, semi-closed and open. The medical problems in connection with each class of compartment will vary considerably.

Open stations would comprise all places such as the fighting tops, anti-aircraft gun platforms, etc., and the protection of the personnel there will necessarily be limited to the use of the mask and protective clothing.

Semi-closed stations would include all such compartments as are necessarily connected with the air without the ship. Among such compartments may be mentioned the conning-tower, turrets, broad-side batteries, battle signal-stations, and the fire and engine-rooms. In these the protection afforded by masks and special clothing will be supplemented when possible by filtration of the air in the plenum ventilating system and the maintenance wherever practicable of a strong positive air pressure sufficient to prevent the entry of gas into the compartment through gun ports or other natural openings.

Under the heading of closed spaces we can consider such compartments as the torpedo room, dynamo room, plotting room and the magazines. By completely sealing off such compartments with gas tight doors, diaphragms on voice tubes, etc., and the installation of compressed air or oxygen tanks and air purifiers the problem of the protection of such spaces from gas, excepting a direct hit by a gas shell is solved. And it seems likely that in the future more and more compartments of the ship will be converted into closed spaces by the introduction of submarine methods of self maintenance as regards air supply (9).

The Medical officer's concern in these arrangements will arise mainly from the necessity of assuring himself that the air is being maintained at the necessary degree of purity, that the humidity or temperature of the compartment is not permitted to pass beyond the specified limits, and that the air within the compartments is kept in motion by fans.

In addition to the above he must assure himself that the food and water supply of the ship is being properly safe guarded. All air con-

SMOKE CURTAIN AS DEMONSTRATED AT NEW YORK CITY HARBOR, MAY 2, 1924

nections of water tanks with the exterior should be protected by some modification of the gas mask canister, and exposed food protected by impervious cloth or paper containers.

If the ship has been subjected, while in harbor or at a navy yard to an airplane attack during which gas been used either by drop bombs or spray the medical officer should regard the harbor water as being contaminated and should satisfy himself that all reasonable means are being employed to prevent the chemical from coming in contact with the personnel of the ship. Such contact would result from the use of harbor water in the heads, for scrubbing the decks, from its use for washing clothes, or by hoisting in boats which have been exposed to contamination. Since many persistent agents are not destroyed for days by exposure to water the danger to the personnel from harbor water contaminated by chemicals is at least as great as that present in a harbor adjacent to a city infected by cholera.

Similarly the medical officer should be prepared to rapidly handle cases from a landing force which has been compelled to attack a beach protected by persistent agents previously sprayed about as a defensive measure.

Another feature of the measures taken for collective protection that will engage the medical officers attention is the training of the members of the first aid and stretcher parties to promptly recognize the gas alarm and to know what doors and gas partitions have been sealed in consequence, and how this will affect the routing of casualties of any kind. Stretcher and first aid men should not allow themselves to be sealed off in small compartments and so lose their availability for use among larger groups of wounded.

Following an attack the active direction of degassing the ship and its personnel will fall upon the gas officer but the medical officer should hold himself in readiness to consult upon all measures taken, as these will so largely affect the future health and safety of the crew. The cleansing of decks, bulkheads, machinery and guns must first be carried out rapidly and effectively, following which the degassing so necessary to maintain the safety of the personnel must be undertaken. If persistent agents have been used clean clothing should be issued and the contaminated clothing degassed by boiling for an hour or destroyed. Food and water exposed should be thrown overboard. One of the first duties of the medical officer following a gas attack will be to assure himself that the scuttle-butts are not contaminated. If so they can be degassed with live steam supplemented by cleaning

with anti-gas compounds, followed by scrubbing with water. The mess gear, tables, benches, hammocks, officers and crews heads having been carefully degassed the medical officer should satisfy himself by personal inspection that the sanitary integrity of the ship has been as far as possible returned to normal.

Most of the degassing substances used are either powerful chlorinating or oxidizing agents. As a result the toxic chemicals they are used upon are converted either to more highly chlorinated compounds, to vinyl derivatives, or are oxidized. The resulting compounds, while harmless to man usually have exceedingly obnoxious odors, and these compounds will have to be thoroughly removed from the compartments before the confidence of the crew can be restored. While the degassing is proceeding the men engaged in it should be carefully supervised to prevent their accidentally coming in contact with heavily gassed objects and first aid supplies should be readily available for the immediate treatment of chemical burns.

In addition to aiding in the execution of the above measures the medical officer should be prepared, with the aid of his stores to establish small degassing stations for gas mask face pieces and other material which cannot be boiled or treated with strong degassing agents. This can usually be accomplished by generating (by means of chloride of lime and hydrochloric acid) a cloud of chlorine in a closed box or tent on deck and placing in it such articles as desired. Delicate instruments liable to corrosion from the gas cannot be exposed in this manner. Practically all immediately necessary sick bay gear can be degassed by boiling or the use of live steam under pressure.

In past wars the medical officer has always made certain preparations to anticipate the dangers to which the personnel of various parts of the ship might be exposed in action, i.e., extra dressings for men on the upper decks exposed to splinters, extra supplies for men in the fire and engine rooms exposed to live steam burns from pipes cut by shell-fire, etc. During a gas attack similar variations will arise in the degree of exposure of different groups, and the percentage of gas casualties will vary accordingly. Those men working in gas-tight compartments will only have to fear the results of a direct hit from a chemical shell. Men in the semi-closed stations are in danger at all times from any form of attack, either cloud or direct hits. Men in the open spaces will be exposed to cloud attacks and splashes from direct hits but their main concern will be with cloud attacks, and

Burst of 100 Pound Phosphorus Bomb

as soon as the ship has steamed out of the cloud the possibility of their becoming casualties would cease. This would not be true of the members of the crew below decks where the chemical agent from the cloud being confined would constitute a menace for some time, the more especially if a persistent agent were used. If after the attack the ventilating system can be run at all full speed the compartments will be degassed of agents present in the vapor form, the length of the period required to degas the compartment varying with its size, rate of air change, and the nature of the agent used and the ship's anti-gas officer will be able to provide the medical officer with figures indicating the probable rate of degassing of all compartments.

From a consideration of all the above factors as applied to his own ship the medical officer can make a fair estimate of the conditions that will confront him during a chemical attack and make his dispositions accordingly.

The shortage of medical personnel and the restriction of space aboard ship makes the establishing of special gas aid stations (as is done in the Army) impracticable, and gassed cases will gravitate to the usual battle dressing stations by the same routes as the wounded. These routes will have to be even more carefully selected in the future than in the past, for in addition to the handicaps already imposed by water-tight doors the introduction of anti-gas doors and screens will make the finding the routes which are always open a difficult problem.

First aid packages for gas poisoning will have to be as simple as possible. The introduction of a large number of chemicals would only lead to confusion in their use during an attack, and as the crew would all be masked and the administration of any eye treatments or internal treatments therefore be impossible, the only chemicals necessary would be those employed against gas burns, such as a 5 per cent solution of potassium hydrate, a 2.5 per cent solution of sodium bicarbonate and a large bottle of carbon tetrachloride with a good supply of gauze sponges for the immediate removal of mustard splashes. In addition to the confusion likely to result from the presence of a large number of chemicals in the first aid boxes there would be an additional danger in that all such bottles might become secondary missiles following a direct hit by a shell.

In naval warfare a great many cases receive head injuries and will be unable to wear the regular mask. It therefore becomes a question as to whether head injury masks should be placed in the gas first aid

boxes in order that head injury cases can be protected during a gas attack. As all the crew are fully occupied during an engagement and unable to spare the time necessary for adjustment of these masks it seems likely that the best solution will be to have two of these masks fastened to each litter or stretcher and the stretchermen can adjust them to such head cases as are unable to wear the ordinary mask. Stretchermen and corpsmen detailed for first aid duties should also be thoroughly conversant with the usual first aid treatment of different gases, and after having been told the nature of the gas attack the ship is undergoing should be alert to see that cases are handled as expeditiously as possible; i.e., in the event of a mustard gas attack they can tell the cases to walk to the battle dressing station whereas men seriously gassed with lung irritants will have to be transported by stretcher.

Upon the arrival of gassed cases at the dressing station the procedure to be followed will vary with the nature of the cases. If the dressing station is protected from gas by gas-tight curtains or doors the cases will have to be undressed outside the doors, especially if persistent agents have been used, in order to prevent contamination of the dressing station. The gassed clothing should be thrown in tubs of weak chloride of lime which will rapidly neutralize most known agents. A medical officer should then examine the cases as quickly as possible to determine how many are fit to return to their action stations immediately. In making these decisions he should return all doubtful cases to their stations at once. For the popular fear of poison gas is such that during the late war many sound men were permitted to filter to the rear upon claiming they had been gassed, and while many of these men undoubtedly had been slightly gassed they were not casualties and should have remained with their units. In naval warfare it is very doubtful if any division officer would permit men to leave their stations unless certain they had been so seriously gassed as to be merely encumbrances rather than of assistance, but the medical officer should nevertheless satisfy himself as to the seriousness of the case and should always hold in mind the fact that with certain gases (such as mustard) no harm is done the patient by exercise, the symptoms and disability only developing after many hours or a day.

Cases obviously unable to do duty should be retained and the course of treatment to be followed will depend upon the agent used and the severity and nature of the case. Chemical burns from

splashes or highly concentrated clouds should be treated at once as outlined elsewhere in this book. Similarly the handling of cases disabled by lachrymators, toxic smokes or lung irritants will depend upon the severity of the case. As a general rule it is believed that nearly all such cases can be laid to one side in a group and palliative treatment administered by a specially trained hospital corpsman until such time as a lull in the stream of wounded permits the medical officer to give each case more attention. Exceptions to the above rule would be men gassed by lung irritants; in these cases early venesection and the injection of gum glucose intravenously will prevent the later development of lung edema. Even these treatments can be administered by a skilled assistant when so directed by the medical officer.

Many cases which on admission appear to be severe will soon recover and can be sent back to their stations in an hour or so. This is especially true of cases that have been lightly gassed by sensory irritants, and who are perfectly able to do duty after the initial symptoms have passed away, for in such lightly gassed cases no serious sequelae are to be feared. Finally it can be stated that as usual each case must be judged on its own merits and treated accordingly.

The supplies required in the battle dressing stations are as follows:

Hospital masks for head injury cases.
Carbon tetrachloride and chloride of lime for the treatment of mustard burns.
A large supply of cheap gauze or cotton sponges for use with the carbon tetrachloride.
Sodium hydroxide solution, 5 per cent.
Sodium bicarbonate solution, 2.5 per cent for use on eye, nose and throat cases.
Ferric hydroxide glycerine paste made as described in Chapter VIII.
The usual morphine solutions and syringes already provided.
Alcohol chloroform and ether mixture for toxic smoke cases (see Chapter X).
Venesection outfit.
Dichloramine-T or Dakin's solution.
Intravenous apparatus with gum-glucose solution for same.

As difficulty will be experienced in hanging the reservoir high enough aboard ship to get the required pressure it may become necessary to do the injections with a large glass syringe connected to a reservoir by a two-way stop-cock.

While the use of oxygen is urgently indicated in many gassed cases its use during an action is contraindicated by the fact that oxygen

actively supports combustion, and any accident to the cylinders would produce a fire-hazard of high degree.

With the aid of the above supplies, plus any remedies found to be of special value against new chemical agents released by the enemy most cases can be carried through the heat of the engagement.

After the battle it becomes necessary to treat any gassed cases more thoroughly, and this will be done along the lines laid down in the previous chapters.

For the later treatment of the lung irritant cases it has been suggested that a compartment aboard could be set aside and used as an oxygen chamber, but this is not considered feasible because of the large amount of oxygen required and the fire-risk involved. If used at all for treatment on a fighting-unit the oxygen should be drawn from a cylinder to ordinary gas piping installed along a bulkhead, this piping to have rubber hose offshoots every 3 feet. Haldane masks, being the most economical as regards oxygen consumption, are attached to these rubber hose-leads and the patients can be placed on cots or stretchers, one below each lead. By using this method one hospital-corpsman can supervise the treatment of many cases with the minimum loss of time, and by keeping these cases grouped together confusion will be avoided.

The later treatment of vesicant cases will consist of frequent dressings of the burns and blistered areas of mustard cases, and the excision, as soon as practicable of the burned areas resulting from splashes of lewisite and similar agents. The development of severe obstructive symptoms in the trachea, following the inhalation of the vesicant gases will call for insufflations and possibly even intratracheal injections through the crico-thyroid membrane.

Following an engagement one of the problems that arises is what cases to transfer to the hospital-ships or hospital. And the same considerations that govern the medical officer in his decisions on wounded cases will influence him in his disposal of the gassed. That is to say, lightly gassed men who by reason of special training or ability are of great value will be retained aboard. However, in his zeal to preserve the integrity of the personnel of the ship the medical officer will probably retain aboard many cases that should be sent ashore, not only for their own benefit but for the benefit of the ship as a fighting unit. Gas burns heal with extraordinary slowness and a burn which at first sight gives promise of healing in a few days will go on to sloughing and refuses to heal under the best treatment.

Eye cases show a photophobia that may last for months and is all out of proportion to any visible lesion. Inhalation cases develop a host of symptoms following their apparent recovery and are totally useless so far as life aboard an actively cruising ship is concerned. And there is no way of telling what cases will develop these symptoms. So whereas the medical officer can often retain lightly wounded cases on board, being secure in his knowledge of the eventual outcome, the principle does not hold true with gassed cases. For while they all finally recover and while the practice of coddling such cases must be avoided, the only place where the graduated exercises and special treatment necessary to return them to normal can be carried out is in a properly equipped hospital with skilled direction.

In the above paragraphs an effort has been made to foresee what would be required of a medical officer on a battleship during a war in which chemical agents are introduced at sea, and to provide a basis for the development of the medical department to meet chemical warfare problems. The same measures would be taken on other large combatant units, such as battle cruisers and plane carriers. Ships of the train while less liable to be exposed to direct hits during an engagement are equally exposed to other forms of attack, such as an attack by planes and their medical departments should be prepared to deal with gassed cases.

As to scout-cruisers, flotilla-leaders and destroyers the conditions resulting from chemical warfare will, in the main, resemble the conditions on major vessels. It seems likely, however, that owing to their light armor the first few shells would convert many of the closed and semi-closed compartments into open compartments communicating directly with the external air and so a large part of the personnel would be exposed to subsequent cloud attacks. On destroyers the bulk of the crew engaged in handling guns and torpedo tubes, and in maneuvering the ship are completely exposed to cloud attacks at all times and the Chief Pharmacists Mate should be fully prepared to render first aid to any casualties. Although on smaller vessels the thorough treatment of a large number of gas casualties is impossible much can be done to make them comfortable and to return the lightly gassed cases to duty at the earliest possible time.

While it is very unlikely that cruising submarines will be much exposed to gas attacks, it is possible that they might be attacked while at anchor. A source of possible danger would lie in the personnel using their submarine masks against war gases, for while the protec-

tion afforded by this mask is very high against chlorine and moderate against phosgene there is absolutely no protection afforded against toxic smokes and only slight protection against the arsenicals such as lewisite and methyldichlorarsine. Moreover the use of this mask against any war gas other than chlorine (which is completely removed by the causticized pumice) will lead to "poisoning" of the Hopcalite in the canister and the mask would be of no further value as a carbon monoxide remover.

Hospital ships while presumably immune to attack might be subjected to gassing in the event of a cloud gas or airplane attack on a fleet in formation or at anchor. For this reason a certain amount of anti-gas preparation will have to be made even aboard these non-combatant ships, and in addition to the usual methods employed aboard all vessels, such as the stopping of ventilation blowers, the closing of all openings such as ports, etc., the hospital corps personnel must be trained to rapidly mask all patients too ill to adjust their own masks. The rapid and orderly execution of all these maneuvers, between the time that the gas alarm is sounded and the gas cloud hits the hospital-ship presupposes a high state of gas discipline, for on sea as on land the best anti-gas devices are of no value in the absence of a personnel thoroughly trained to their use.

As regards additional medical stores to be carried by a hospital-ship in anticipation of the receipt of the gas casualties from a fleet it is believed that the stores carried at present with the addition of a large number of oxygen cylinders and Haldane oxygen masks will meet the requirements. These supplies when supplemented by such special remedies as may be found to be of value in the future should meet all requirements.

The installation of oxygen chambers aboard would introduce a fire-risk too great to be tolerated and such chambers are of more value in treating the late polycythemias, nocturnal dyspneas and neuroses of old gassed cases and so would only be of value in shore hospitals.

In view of the British and French experience during the World War during which they finally collected their gassed cases in special hospitals it would seem advisable for all Navy gas convalescents to be sent to one hospital where with special facilities and specially trained personnel their recovery could be hastened. The hospital selected should be one located in an equable climate.

DANGERS OF TOXIC GASES

While the main concern of the medical officer with the more toxic gases arises from their possible employment by an unscrupulous enemy during a naval war, occasions may arise at any time which will demand his professional aid in combating gas emergencies. Toxic gases are being employed more every year for various industrial purposes, and their use has been gradually extended to many marine activities. As examples may be cited the introduction of hydrocyanic acid and cyanogen chloride in ship fumigation, the use of ammonia in cold storage plants and the introduction of new fuels for motors. In addition to these sources of danger toxic gases may be generated aboard ship owing to defective apparatus, as a result of the decomposition of usually stable chemicals or as products of explosions and fires.

A number of marine disasters have occurred from the preceding causes and the medical officer should be cognizant of the mode of action of the gases concerned, the diseases resulting from exposure to them and the defensive measures to be adopted in preventing further casualties.

Ammonia

Many merchant ships are equipped with cold storage plants chilled by the use of ammonia. Some of the Army transports are similarly equipped and in consequence a certain number of ammonia canisters are issued to transports for use in emergency or during repair work on the ice-making machines. Ammonia fumes are highly dangerous and a break in the line in the confined spaces aboard ship may cause a number of serious casualties, such as have occurred in ice-plant accidents in many cities. When inhaled, ammonia can be detected when only 0.005 per cent is present in air, the eyes are irritated by 0.07 per cent and coughing is produced by 0.17 per cent (American University experimental station report). Lehmann (13) states it is lethal in concentration of from 1.74 to 3.13 mgm. per liter for half or one hour exposures which gives it about the same toxicity as chlorine. Its toxic effects are due entirely to its local irritant action on the respiratory organs. Magnus (14) and Sihle (15) have shown that the lungs are impervious to this gas and it is neither absorbed into the blood nor eliminated from it in the lungs. The symptoms are a sense of anxiety, pain in the epigastrium, giddiness, burning in the throat,

and vomiting. The pulse is small and frequent, the mouth and throat reddened, with increased secretion. In animal experiments there is increased secretion of eyes, nose and mouth, remarkable hoarseness, and vomiting followed by tetanic convulsions and death. Blyth (16) states that "on section, the bronchial tubes, to the finest ramifications are found to be filled with a tenacious mucus, and the air passages, from the glottis throughout, reddened. The lungs are emphysematous, but have not always any special color; the heart contains but little coagulated blood; the blood has a dark red color."

Cushny (17) states that the irritation of the nasal mucous membrane causes a reflex vaso-constriction with increased blood-pressure, the heart is temporarily slowed. The respiration is first arrested, and then becomes deeper and fuller.

A concentration of 3 mgm. per liter causes marked and immediate skin irritation.

Men working around ammonia plants often develop chronic conjunctivitis and skin changes, the latter probably due to the ammonia combining with the oil of the skin glands to form irritant soaps.

Skin burns with ammonia are best treated with the usual picric acid solution; the eyes can be treated with a saturated solution of boric acid. When ammonia has been inhaled a cloth dipped in vinegar and layed over the mouth and nose gives relief (18).

The Army and Navy mask with the blue canister gives only three minutes protection against a concentration of 3.5 mgm. per liter. With the green (training) canister, only two minutes protection is given against the same concentration. It can be seen that the present gas mask canister would be of no value in case of accident. In order to meet this danger on board army transports the Chemical Warfare Service has developed an ammonia canister. This consists of an extra large canister filled with 1100 cc. of pumice impregnated in a concentrated copper sulphate solution. This canister will give protection for over one hour against a concentration of 3.5 mgm. per liter of NH_3. The Navy carbon monoxide mask gives no protection against ammonia. As ammonia is very soluble in water a cloth soaked in water and tied over the mouth and nose gives some protection. Similarly, water sprayed on a leak in the ammonia line will reduce the concentration greatly.

Hydrogen cyanide and cyanogen chloride

Until recently ships were fumigated with either sulphur dioxide or hydrocyanic acid. Either method had numerous disadvantages which led to the adoption by the Public Health Service of the gas now used, a mixture of hydrogen cyanide and cyanogen chloride. This gas is generated by dropping bags of sodium cyanide, sodium chlorate and talc into crocks containing hydrochloric acid. In the proportions used at present the most concentrated gas air mixture (that used for the destruction of lice, bed-bugs, and roaches) set up in a compartment would result in a concentration of 1.88 ounces per 1000 cubic feet (1.882 mgm. per liter) of cyanogen chloride and 0.654 ounces per 1000 cubic feet (0.654 mgm. per liter) of hydrogen cyanide. Such a mixture, the strongest used in fumigation is lethal to rodents in a few minutes and the irritant effects of the cyanogen chloride make it intolerable to the eyes and nose. One-sixteenth of the above concentrations is non-lethal to rodents for an hour's exposure but is intensely irritating to man. The presence of the irritant cyanogen chloride makes this gas an excellent fumigant, combining as it does the toxic effect of the cyanogen group with the warning effect on operators of the cyanogen chloride.

The effects of hydrocyanic acid have been considered in Chapter XI, the effects of cyanogen chloride are as follows: Concentrations of 0.0025 mgm. per liter produce marked lachrymation in a few minutes, and a concentration of 0.005 mgm. per liter causes irritation of the throat and nose in addition to lachrymation. The lethal concentration for dogs, two hundred and forty minutes exposure, is 0.12 mgm. per liter so it can be seen that there is a large margin of safety between the concentration at which the gas gives warning of its presence and its lethal concentration. Cyanogen chloride kills (as do practically all cyanides) by virtue of the cyanogen group it contains and therefore the symptoms of poisoning are similar to those of hydrocyanic acid (Chapter XI), with the addition of immediate excitement and irritation of the eyes and respiratory tract. The pathology of animals gassed by cyanogen chloride differs from that of hydrocyanic acid in that there is considerable irritant action by the halogen compound on the respiratory tract, with desquamation of the epithelium of the trachea and bronchi, a mild inflammatory reaction around the small bronchioles, perivascular edema, and slight hemorrhage and edema in the alveoli.

Long exposures to low concentrations produce only a slight conjunctivitis, hoarseness and occasionally digestive disturbances.

Treatment of acute cases is precisely the same as for hydrocyanic acid poisoning, artificial respiration in pure air.

If for any reason it becomes necessary to enter compartments while the fumigant is still present the various gas masks give protection as follows.

The Public Health canister (which is painted white) contains 750 cc. of charcoal impregnated with 2 per cent hexamethylenetetramine solution and 400 cc. of causticized pumice and will give five to six hours protection against a mixture of 1.02 mgm. per liter of cyanogen chloride and 0.47 mgm. per liter of hydrocyanic acid, and thirty minutes protection against a concentration of CNCl 5 mgm. per liter and HCN 1.7 mgm. per liter. These high concentrations give early warning of their presence by redness and itching of the skin and the white canister gives ample protection against them.

The 1919 blue canister as used by the Navy and the green canister issued for training purposes give good protection against the fumigant mixture, the blue protecting against a concentration of HCN of 2.2 mgm. per liter for thirteen minutes, and a concentration of CNCl of 2.5 mgm. per liter for thirty minutes. The green canister gives the following protection.

	MILLIGRAMS PER LITER	MINUTES
Hydrocyanic acid	0.55	29
Hydrocyanic acid	2.2	16
Cyanogen chloride	0.25	10
Cyanogen chloride	1.25	5

From the above figures it can be seen that the navy gas masks can be used in rescuing personnel from compartments filled with the ordinary fumigant mixture, and as the cyanogen chloride breaks through first, warning will be given by lachrymation of the beginning failure of the canister.

The Navy submarine mask will give protection against the ordinary fumigant concentration for twenty-five minutes.

Formaldehyde, HCHO

Formaldehyde vapor is often used on board ship for disinfecting purposes. Exposure to low concentrations causes irritation of all

mucous surfaces, but a certain amount of tolerance is developed by those constantly exposed to the vapors. High concentrations are very irritant, causing bronchitis and broncho-pneumonia. A portion of the formaldehyde inhaled is absorbed from the lungs and is oxidized in the tissues, especially the liver, to formic acid, and a part is converted to hexamethylenamin. The remainder is destroyed in the body or excreted as formaldehyde.

The Manual for the Medical Department, United States Navy, 1922, gives directions for the use of formaldehyde which would result in the establishing of a concentration of 4 mgm. per liter in compartments and rooms. As the green canister protects against a concentration of 12.27 mgm. per liter for over forty minutes, and the blue canister against a concentration of 30 mgm. per liter for over forty minutes these masks can be used in rooms filled with formaldehyde vapor. The Navy submarine mask gives absolutely no protection against formaldehyde vapor.

Nitrous fumes, NO_2

The effects of nitrous fumes have been considered in Chapter XI. They are very likely to be met with in burning chemical plants where nitric acid or certain nitrates are stored. Their principal interest to the medical officer arises from their being generated whenever the nitro powders burn slowly or are incompletely detonated. The great bulk of gassed cases in the British Navy during the World War were due to nitrous fumes and carbon-monoxide. The fumes of nitrous oxide are reddish brown in color and in the presence of moisture rapidly change to nitrous and nitric acid which is probably the cause of the irritant effects of these gases. Haldane (19) found that mice exposed for half an hour to a concentration of 0.05 per cent (1.88 mgm. per liter) died with the typical symptoms in twenty-four hours.

Symons (20) reports

> Early in the morning of April 27, H. M. S. *Russell* was struck by two mines, and the after part of the ship caught fire. Later an explosion occurred in the after turret. It is certain that in the after part of the ship were large quantities of fumes from burning explosives, chiefly cordite, and that many officers and men inhaled these fumes. Seventeen officers and men were subsequently treated in the Royal Naval Hospital, Malta, suffering from poisoning from some of these gases.

Symons showed that these 17 cases were due to nitrous fumes. Fourteen of them terminated fatally.

Fairlie (21) reports the results of the torpedoing of H. M. S. *Britannia* on November 9, 1918. Following the explosion of the torpedo a second explosion seems to have occurred and also a fire in one of the magazines with the result that burning cordite was present. Several gassed cases died on relief ships and 2 on their way to the hospital. Fifty-six gassed cases were admitted to hospital, 10 of whom died. Some of the exposures to the gas were only of two to five minutes, and yet terminated fatally.

A prolonged latent period between the period of exposure and the onset of serious symptoms was noted, many cases only developing after eighteen hours.

The cases all ran the usual course of pulmonary edema found in nitric oxide poisoning.

Fairlie found emetics and atropine to be useful in the early stages, but not in the later. Bleeding was of great value. Where the blood was very viscid, venesection of the median basilic or median cephalic was performed. Strict diet, rest, warmth and stimulants were necessary. Oxygen was found of little value, undoubtedly because no proper masks were at hand as Fairlie points out.

The blue or green canister protects for at least seventy minutes against a concentration of 5.64 mgm. per liter. The Navy submarine mask will protect one half hour against a concentration of 3.76 mgm. per liter.

Fire extinguisher fumes

Many fatal accidents in industrial plants, tunnels and aboard ship have resulted from the use in confined spaces of certain fire extinguisher fluids. Practically all of these fluids consist of carbon tetrachloride with an admixture of chloroform or a high boiling petroleum distillate to prevent freezing. Fieldner, Katz, and Kinney (22) have investigated the behavior of various fire extinguishing liquids in a 1000 cubic foot chamber by spraying the fluid on fires of different materials and also on red hot channel irons. Gas air samples were then drawn and analyzed. The results showed that carbon monoxide was given off in small amounts, not sufficient to be dangerous during the brief exposure of an operation. Chlorine was generated in very small amounts and hydrochloric acid vapor was evolved in amounts sufficient to be irritating but not dangerous. Carbon tetrachloride

vapor was present in concentrations ranging from 0.2 to 0.6 per cent, which is not sufficient to produce anesthesia in a short time but longer exposures would cause sudden unconsciousness. Phosgene was given off in concentrations, varying with the conditions, of from 15 parts per million (0.060 mgm. per liter) with excelsior fires to 168 parts per million (0.680 mgm. per liter) with heated iron, the latter concentration being lethal. The yellow color of the smoke produced is due to the formation of iron chloride which hydrolizes to hydrochloric acid and iron oxide.

The blue or green canisters give perfect protection against any of the gases evolved except carbon monoxide which is present in such small quantities as not to be dangerous during a brief period. While these fire-extinguishers are of great value, especially in incipient fires, great care should be exercised in their use in compartments without adequate ventilation and where rapid escape is impossible.

Sulphur dioxide

This gas is often used on board ship as a fumigant. It is a colorless irrespirable gas with the odor of brimstone, is heavier than air and is condensed to a liquid at −18°C. Siphons of this liquid are sold for fumigating purposes, but in marine use the general practice is to burn sulphur sticks or lumps. It was much used in medieval chemical attacks to suffocate the enemy. Sulphur dioxide readily oxidizes to sulphur trioxide which in turn combines with water to form sulphuric acid, and many men have been burned by coming in contact with the interior of the ship's funnels after fuels with high sulphur contents have been used.

When inhaled, intolerably irritant effects are at once experienced from a concentration of 5.72 mgm. per liter and coughing and sneezing result. In fatal cases the post-mortem appearance is that of asphyxia. The blood is very dark, the bronchi and bronchioles present a croupous appearance. The lungs show edema. Mild alkalis have been given as antidotes on the theory that they combat the acid produced.

A cloth or sponge soaked in water and fastened over the face gives some protection.

The green canister protects against a concentration of 131 mgm. per liter for twenty-nine minutes, the blue for at least as long. The submarine mask will protect an hour against 0.9 mgm. per liter. Since the highest concentration used in fumigating is 128 mgm. per

liter (produced by burning four pounds of sulphur in a space of 1000 cubic feet) it can be seen that the blue and green canister give adequate protection for short exposures and the submarine mask is of no value.

Hydrogen sulphide, H_2S

This gas is highly toxic to men, 3 mgm. to the liter causing death in two to three minutes (23). It was used by the British in several cylinder attacks but abandoned because of its inflammability. A large number of fatal accidents have occurred in sewers and industrial plants as a result of the inhalation of hydrogen sulphide and these accidents are not infrequent aboard ships where organic matter has been allowed to decay as occurred on the U. S. S. *Wyoming* (24).

In the forward part of the ship, below the space occupied by the wardroom, there is a sump tank into which drains from the torpedo room lead. Soap and other alkaline cleansing agents along with water and oils and grease used in the care and preservation of the torpedoes were washed into this tank which had to be flushed out and cleaned periodically. Whenever the manhole plate which was situated at the bottom of a trunk was removed a strong odor of hydrogen sulphide permeated through the forward sections of the ship up to the upper deck. The tank was not adequately vented, but apart from that it appeared that the gas was evolved from action by the alkaline cleansing agents upon the oils used in the torpedo room.

Upon one occasion when the tank was opened for cleansing, the man who removed the manhole plate was promptly overcome and was carried to the sick bay unconscious. For an hour or so it looked as though he might die. He was partially conscious but his skin was cold and gray with a peculiar yellowish tinge on the face. The pulse was rapid, small, and soft and his breathing shallow. He was surrounded with hot water bags and blankets and stimulated with coffee, strychnine, and adrenalin chloride. The following day he was able to be up and about but was still weak. Three other men who were in the trunk were affected but not sufficiently to render them unfit for work. They vomited and complained of muscular weakness and headache.

For several hours after the manhole plate was removed the odor of hydrogen sulphide was strong in the wardroom and wardroom country. The white paint work in passageways between magazines in the vicinity of the trunk through which the gas escaped turned dark yellow, and bright metal surfaces were affected as might be expected.

Inhalation of large quantities causes cyanosis, loss of consciousness and frothing at the mouth with ridigity of the muscles, and septic pneumonia may follow. Many theories as to the mode of action of hydrogen sulphide have been advanced, none of them are satisfactory. It seems likely that in high concentrations it is directly toxic to the

nervous system. In chronic cases headache, conjunctivitis and gastric disturbances are common.

The green canister protects against hydrogen sulphide 13.91 mgm. per liter for ninety-seven minutes. The blue canister protects for one hundred and twenty-five minutes against the same concentration. The submarine canister gives no protection.

Chlorine and carbon monoxide

These two gases have been considered elsewhere in this book. Their principal cause of interest to the medical officer at sea arises from the fact that both these gases are occasionally liberated in submarines by defective machinery or accidents. The Navy submarine mask was designed to give protection against these two gases. As the air passing through the mask is absolutely dry, complaint may be made of dryness of the throat and nose, but as the masks are only for emergencies this defect does not interfere with their use.

The usual method of testing air in mines or compartments for the presence of CO is to introduce a canary bird and watch for toxic symptoms. A more delicate method, and one more applicable to ship conditions is that described by Hoover (25).

The detector material is composed of a mixture of 10.6 per cent iodine pentoxide, 33 per cent pumice stone of 8 to 14 mesh (or some such mesh), and 56 per cent fuming sulphuric acid which contains 60 per cent sulphur trioxide. The material is kept dry. The detector is put in a tube of 5 mm. diameter to a depth of 5 mm. The air is pumped through the detector by squeezing a bulb attached to the tube. The air passes through a layer of activated charcoal before entering the detector. The detector is sufficiently sensitive to detect in a few seconds quantities of carbon monoxide less than that which is harmful to man. A green color is developed in the detector in the presence of carbon monoxide. A permanent color standard made of basic copper acetate and chromium oxide may be prepared for comparison with the color developed. Hoover claims that by pumping 500 cc. of air through the tube a concentration of 0.005 per cent can be detected.

Phosphine, PH_3

This is a colorless, inflammable highly poisonous gas. It causes dyspnea, a sense of pain and pressure in the chest and asphyxia. At autopsy the lungs are found to be congested and edematous with

capillary hemorrhages. Its interest to the medical officer arises from the fact that fatal poisonings by inhalation have occurred on many ships carrying ferrosilicium as cargo. This substance is a mixture of iron ore, quartz, coke and lime, which in the presence of moisture gives off vapors containing 95 per cent of phosphine.

Excellent protection is given by both the green and blue canisters against phosphine. No protection is given by the submarine mask.

Gasoline fumes

Cases of gasoline poisoning occasionally occur aboard ship from exposure to the fumes. The first symptoms are headache, giddiness and inebriation. In high concentrations muscular tremors, convulsions, unconsciousness, dyspnea, cyanosis and death may follow. In animals an anesthetic effect has been noted. Autopsy showed a marked congestion of the lungs and a bloody mucus covering the mucosa of the trachea and bronchi. Similar symptoms and death have also followed the inhalation of turpentine in small compartments. The treatment is symptomatic.

The green canister protects against gasoline, 4000 parts per million for fifteen minutes. The blue canisters protects against the same concentration for thirty-five minutes. The protection against turpentine is even greater. The Navy submarine mask gives no protection against either vapor.

Arsine, AsH_3

The effects of arsine have already been considered in Chapter XI. On several occasions arsine has been developed aboard submarines, the resulting cases differing markedly from the usual picture of arsenic poisoning. A large number of cases occurring on the British submarines D 3 and D 4 are described by Dudley (26). In June, 1916, both these boats took trial trips of from four to eight days duration during which trips they were submerged on an average of seventeen hours a day. The presence of defective battery grids which contained a high percentage of arsenic led to the development of arsine in the submarines. Vomiting and dyspnea were the commonest complaints among the crews, developing early after exposure. Hemoglobinuria and jaundice were constant signs, edema of the face and eyelids occurred in many. Headache and albuminuria were common. All of the cases except four developed mild symptoms of a neuritic character. The red cells were reduced in every case, in one case to 1,780,000;

the color index was first high, and then as recovery occurred was slightly below 1. Evidence was found of an increase in fragility of the red corpuscles. Arsenic was recovered from the urine, the hair, and the finger and toe-nails. Marked evidence of idiosyncracy to a poison was shown, the cases varying markedly in degree although the arsine was uniformly distributed throughout the submarines. One man claimed to have no ill effects from the trips taken. There was no mortality among the total of 56 cases and all had recovered without treatment in six weeks.

Both the blue and green canisters give excellent protection against arsine, but the submarine canister gives no protection.

Lack of oxygen

In the foregoing paragraphs some of the common gases encountered aboard ship are considered. But very often when men are overcome in compartments or holds the principal cause is a lack of oxygen in addition to the presence of toxic gases. Compartments in which food supplies have been stored for some time may show a marked decrease in oxygen content. Brooke (27) describes the accident on board the S. S. *Jacob* carrying a cargo of oranges. Two men entered the hold and immediately fell insensible, and when finally rescued were found to be dead. Analysis of the air in the hold showed

	per cent
CO_2	22.55
O	1.86
N	75.29
CH_4	0.30

Belli (27) by experiments in the Italian navy has shown that the oxygen in a ship's double bottom can be reduced to three per cent by the use of red-lead paint.

Decomposing organic matter in holds or compartments also leads to a reduction in the oxygen percentage. The following is a case in point (12):

> The S. S. *Arabic* was wrecked and sunk close to Boulogne, but was subsequently salved and brought into the harbour. Later on a salvage party was set to work to clear out the grain with which the ship had been laden. The work had been carried out for some time without accident, when one day the working party immediately on descending into the hold were overcome without previous warning by dizziness, failure of vision, loss of power in the arms and legs and loss of consciousness. It was possible to drag some of the

men rapidly from the hold, when they quickly recovered, and no further symptoms developed later on, though some had headache for a time; but 3 men died before they could be rescued. At first it was suggested that carbon monoxide might be responsible for the effects, but there was no source from which it could have arisen and the blood taken from the fatal cases showed no trace of this poison. The symptoms were exactly what would be expected if the men had entered an atmosphere in which the oxygen was greatly reduced. There is little doubt that in this case the sodden and decomposing grain had absorbed a great proportion of the oxygen from the air in the hold. Other gases might have been formed, but if so they can have played but little part. None of the survivors showed any symptoms suggestive of sulphuretted hydrogen poisoning, nor had they experienced on this or on any of the previous days the panting which one would associate with the presence of a material quantity of carbon dioxide. Once or twice previously they had noticed dizziness and weakness of the limbs whilst at work, these symptoms disappearing rapidly if they stopped work for a few minutes, a fact which suggests that on the day of the fatal accident the natural ventilation of the hold was worse than usual.

Naturally none of the gas masks will protect against such a lack of oxygen. In the absence of mine rescue apparatus the light diving helmet can be used. The Army gas mask face piece with a long hose attached and led out to pure air will permit the wearer to enter any atmosphere. Such an arrangement was largely used during the World War when troops desired to explore old abandoned dug-outs and caves in which they had reason to fear a lack of oxygen or overpowering concentrations of poison gas. It use is especially indicated on board ship, where the distance to be travelled by the masked individual can never be very great.

REFERENCES

(1) Thucydides, VII, 43.
(2) Diodorus Siculus, XX, 88.
(3) Groot, Albert: DeMirabilibus, circ. 1350.
(4) Hine, Lt. Col.: Gunpowder and Ammunition.
(5) American Official History of War of Rebellion, Series 1, vol. XXVIII, Pt. 1, p. 33.
(6) McCutcheon, James: Journal Royal Naval Medical Service, vol. V, no. 1, January, 1919, p. 9.
(7) Beebe, C. H.: Chemical Warfare, vol. 9, no. 9, p. 2-4.
(8) Schwarte, M.: Die Technik im Weltkriege. Berlin, 1920, p. 303.
(9) Hains, Paul: United States Naval Institute Proceedings, July, 1923.
(10) Fries and West: Chemical Warfare. New York, 1921.
(11) Jones, Ernest: International Aeronautics, September, 1923.
(12) British Medical History of the War, vol. II, p. 301.

(13) Lehmann, K. B.: Table of poisonous gases in Kobert's Praktische Toxikolgie. Stuttgart, 1912, p. 45.
(14) Magnus: Arch. f. exp. P. u. P., 1902, XLVIII, 100.
(15) Sihle: Zentbl. f. Physiol., 1903–1904, XVII, 238.
(16) Blyth: Poisons, Their Effects and Detection. London, 1920.
(17) Cushny: Pharmacology and Therapeutics. New York, 1918.
(18) National Ammonia Co., New York.
(19) Irvine: Gassing accidents: fumes of explosives. Brit. Med. Journ., January, 1916.
(20) Symons: Poisoning by gases from explosives. Jour. Royal Naval Medical Service, October, 1918.
(21) Fairlie, W. M.: Poisoning by nitrous gases. Jour. Royal Naval Medical Service, January, 1920.
(22) Fieldner, Katz and Kinney: Department of Interior, Bureau of Mines, Tech. Paper 248.
(23) Chemical Warfare Glossary, p. 66.
(24) United States Naval Medical Bulletin, April, 1924.
(25) Hoover, C. R.: Jour. Ind. and Eng. Chem., vol. XIII, p. 770.
(26) Dudley, S. F.: Arseniuretted hydrogen poisoning in submarines. Jour. Royal Naval Medical Service, July, 1919.
(27) Brooke, I. E.: Marine Hygiene and Sanitation. New York, 1920.

CHAPTER XVII

The Treatment of Certain Respiratory Diseases by Chlorine

One of the first things heard on coming to Edgewood Arsenal was the statement that, during the war when the chlorine plant was operating, the workers in this plant did not suffer from colds or influenza. During the influenza epidemic when the hospital was filled with cases, there were practically no admissions from the operatives of the chlorine plant. This statement was confirmed by several intelligent technical men who were at Edgewood Arsenal during the influenza epidemic. One of them added that this immunity only lasted while they worked in the plant, because several of these operatives that were detailed to the hospital as volunteer nurses contracted influenza. Later it was heard from various sources, that operatives in chlorine plants in other parts of the country had always enjoyed a singular immunity to common colds.

The use of chlorine in the treatment of respiratory infections was not new. Hale had used chlorine at the University of Arkansas during the influenza epidemic and believed that students who took this treatment suffered less from influenza than did the general student body. However no systematic study of this method of treatment had been made, and it was determined that this should be the first work of the newly organized Medical Research Division.

With the assistance of Captain H. P. Sawyer, M.C., the concentration of chlorine in the air required to kill certain bacteria on plates was determined. Briefly, it was found, that under proper conditions, a concentration of 0.021 mgm. per liter of chlorine would kill many bacteria in from one to two hours. This statement must not be misinterpreted to mean that this concentration of chlorine will kill bacteria floating in the air. As a matter of fact it will not kill bacteria even on agar plates unless the agar is very moist. It was believed that when a constant stream of chlorinated air was passed over such a film of moisture, that the water absorbed sufficient chlorine to become actively germicidal. Most water supplies are sterilized by the addition of from 0.2 to 0.5 part per million of chlorine, and one part per million of chlorine is usually sufficient even in the presence

of moderate amounts of organic matter. Since these investigations were merely preliminary, and have been reported elsewhere, further details need not be given.

The effect of inhaling chlorine on the bacteria of the naso-pharynx was then tested. Swabs from the naso-pharynx were smeared over the surface of an agar plate. The patient then inhaled chlorine of a

Total cases treated; number of cured, improved and unimproved, with percentage for each

DIAGNOSIS	NUMBER OF CASES	CURED		IMPROVED		NO CHANGE	
		Number	per cent	Number	per cent	Number	per cent
Coryza	388	288	74.2	91	23.5	9	2.3
Acute laryngitis and pharyngitis	127	99	78.0	24	19.0	4	3.1
Acute bronchitis	241	192	80.0	47	19.5	2	0.5
Chronic rhinitis	106	33	31.1	41	38.6	32	30.2
Chronic laryngitis	47	34	72.3	12	25.5	1	2.1
Whooping cough	2	2	100.0				
Influenza	11	9	81.8	2	18.1		
	931	665	71.4	218	23.4	48	5.1

Number of patients taking one, two, three or four one-hour treatments

DIAGNOSIS	CURED				IMPROVED				NO CHANGE 1 HOUR
	1	2	3	4	1	2	3	4	
Coryza	271	16	1		91				9
Acute laryngitis and pharyngitis	93	6			23			1	4
Acute bronchitis	171	14	7		40		7		2
Chronic rhinitis		1	4	28	30	4		6	32
Chronic bronchitis	6	23	5		9		3		1
Chronic laryngitis		1	1						
Whooping cough			2	6		1			
Influenza	6	3			2				
Totals	547	64	20	34	195	5	10	7	48

concentration of approximately 0.02 mgm. per liter for one hour. This concentration is as strong as can safely be borne. In most cases abundant growth was secured on the first plate, while the second culture obtained after exposure to chlorine remained sterile or at most only developed a few scattered colonies. From this it was

believed that an hour's exposure to this concentration of chlorine would practically sterilize the mucous surfaces, and that the undoubtedly excellent therapeutic results obtained later were due to this sterilizing action.

Undoubtedly this conclusion was at least partially erroneous, and later investigations have shown that sterility of the mucous surfaces following the therapeutic administration of chlorine cannot be expected. The error was probably due to two conditions of the experiment. In the first place normal volunteers were used for this work and only the naso-pharynx was swabbed. The normal naso-pharynx gave relatively few bacteria, and sterile plates were obtained after treatment with relative ease. In the second place, a higher concentration of chlorine (0.02 mgm. per liter) was used than was subsequently applied for therapeutic purposes, (0.015 mgm. per liter).

For the treatment of patients an air tight chamber 13 by 13 by 10 feet was constructed, through which 42,000 liters of gas air mixture could be passed per minute, with an even distribution in the chamber. A concentration of chlorine of approximately 0.015 mgm. per liter of air was used, and the concentration was carefully controlled by chemical analyses of the air. The patients remained in the chamber one hour. Many patients were cured by a single treatment, but in many cases a second or third treatment was required on successive days. The results on page 295 were reported March 8, 1924.

Whatever may be the explanation of the efficacy of this treatment, it may be stated here that this gassing chamber has remained in continuous use, and that the results have continued to be approximately the same as those at first reported. The cases treated during the month of June, 1924, may be tabulated as follows:

	CASES	CURED	IMPROVED	NO CHANGE
Coryza	85	60	22	3
Acute bronchitis	9	6	3	0
Acute laryngitis	6	2	4	0
Chronic bronchitis	12	2*	10†	0
Whooping cough	21	4	10	7

*Four months duration.

†Four months to 10 years.

We may now consider further the method by which inhalation of chlorine achieves these results.

1. Although sterilization of the mucous surfaces is not achieved, there can be no doubt that many bacteria are killed. The following table gives the result of swabs taken before and after treatment from ten cases taken at random from among coryza patients treated during the month of June, all that were cultured.

CASE	COLONY COUNT BEFORE TREATMENT	COLONY COUNT AFTER TREATMENT
1	Uncountable	Uncountable
2	30	7
3	Uncountable	8
4	10	11
5	202	7
6	193	78
7	642	340
8	125	38
9	214	41
10	2500	500

We do not know with certainty the organisms that are responsible for the production of colds or influenza, and the Bordet-Gengou bacillus is known to be exceedingly sensitive to unfavorable agencies. It is at least possible that these pathogenic organisms may be killed even though all the common bacteria that grow on our agar plates are not killed. It also seems highly probable that such organisms as remain on the mucous surfaces are much reduced in vitality and virulence even though they may still grow on an agar plate. With many organisms killed and the remainder reduced in virulence, the ordinary defense mechanism of the body may be now able to deal with the remaining infecting organisms.

In addition to disinfectant action by chlorination, "chlorine is a powerful oxidizing agent, and possesses in high degree the property of decomposing toxins. By the regulated action of hypochlorous acid, Dean has prepared a non-toxic dysentery vaccine, and it is a common observation that the free use of hypochlorites may reduce the constitutional symptoms arising from septic processes" (Dakin and Dunham).

Indeed this oxidation is believed by many chemists to be the main disinfectant action of chlorine. Hypochlorous acid is formed by union with water. This in turn is reduced to hydrochloric acid with liberation of nascent oxygen.

It has been suggested that no germicidal action of chlorine could be expected in the presence of the organic matter of the mucus and exudate lining the respiratory tract. This argument loses sight of the fact that it is the great virtue of the chlorinating antiseptics to act under precisely these circumstances. This has been shown by Dakin and Dunham and it is well known that Dakin's solution will cause complete sterilization of a wound, in spite of the very high organic content of wound secretions.

Estimating the tidal air at 500 cc. per respiration, and the number of respirations per minute at 18, 540,000 cc. of air are inhaled and exhaled during one hour. Since a concentration of 0.015 mgm. per liter is approximately equal to 5 parts per million, and since practically no chlorine inhaled is exhaled, about 2.5 cc. of pure gaseous chlorine is absorbed in the respiratory tract during an hour treatment. The thin film of fluid bathing the respiratory tract if gathered together, could hardly be in excess of 50 cc. and hence during the course of the hour this film of fluid receives gaseous chlorine which may be estimated at 5 per cent. To be sure this amount of chlorine is not all present at any one time; but it requires more imagination to suppose that this amount of chlorine has no antiseptic action than to suppose it has.

2. The irritant action of chlorine stimulates the flow of lymph and cleanses the mucous surfaces. One of the first actions of the chlorine treatment is to noticeably increase nasal and bronchial secretion, causing productive coughing and blowing of the nose. When horses were treated with chlorine it was noticed that there was an almost continuous nasal drip, and when they blew their noses or sneezed they scattered discharge all over the clothes of any one standing in front. This cleansing action must necessarily be beneficial, and must aid in disposing of quantities of organisms and may even bring up organisms from the depth of glands and crypts. The fact that sterile plates are seldom secured after treatment may well be due to this continuous discharge of bacteria which have not remained long enough in chlorinated fluid to be killed. Part of the beneficial action in cases of whooping cough is undoubtedly to be attributed to the loosening of the masses of tenacious mucus which are then coughed up more readily, and to this cleansing of the mucous surfaces.

3. The action of any irritant, including chlorine, on the capillaries is to cause a primary brief constriction followed by a much longer

dilatation. Thus a hyperaemia of the affected mucous membranes is produced, which as Bier showed many years ago is conducive to recovery. Not only is such an increased blood supply unfavorable *per se* to the infecting organisms, but such dilatation of the capillaries is invariably followed by an increase in the number of polymorphonuclear leucocytes supplied to the parts which cling to the vessel walls and escape by diapedesis. Thus an increased number of phagocytes are brought to the infected mucous membranes.

4. Stimulation of phagocytosis and the production of antibodies. There are a number of experiments which indicate that physiologically active oxygen stimulates immunological processes. Thus Loevenhart in the 1914 Harvey Lecture, said,

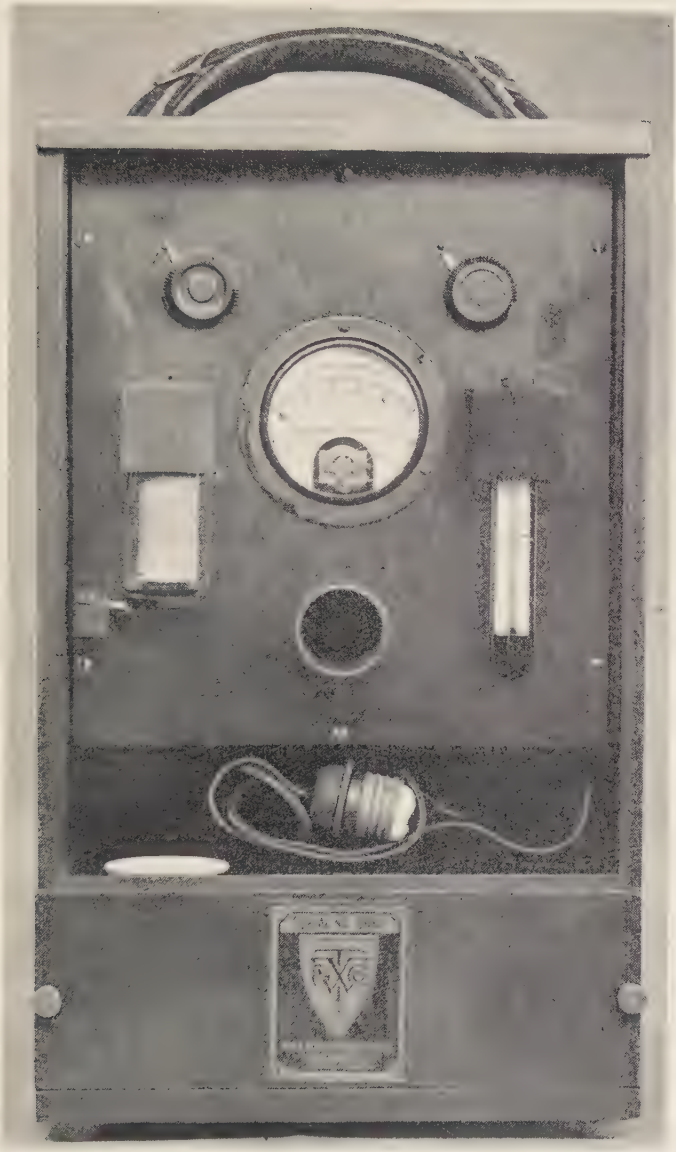
> To return to the original subject of the effect of physiologically active oxygen on inflammation and on immunity processes, I should like to refer briefly to two pieces of work. Prof. L. Hektoen has investigated the effect of iodosobenzoate and iodoxybenzoate on certain immunity reactions. He injected these substances intravenously in dogs and found that they increase the production of specific hemolysin following a single injection of 10 per cent suspension of goat blood. The results were striking. The increase in hemolysin in the animals receiving the treatment was from 12 to 60 times as great as in the control animals. Iodbenzoate without active oxygen is without effect. Whether physiologically active oxygen will similarly stimulate the production of other immune bodies remains to be determined by further experiment. It is sufficiently obvious that if we could similarly increase the production of diptheria antitoxin, it would be of great value.
>
> Dr. S. Amberg and his co-workers in a series of investigations have shown that iodosobenzoate and iodoxybenzoate injected intravenously or intraperitoneally have the power of markedly inhibiting the local inflammatory reaction as produced by mustard oil or bacterial toxins injected intracutaneously or instilled into the conjunctival sac, whereas iodbenzoate, possessing no physiologically active oxygen, is entirely without effect. The results are very striking indeed.

It has also been found that these oxidizing substances stimulate phagocytosis. Adding the power of destroying both exo- and endotoxins to the property of stimulating phagocytosis and antibodies, and it is evident that chlorine may be a powerful therapeutic agent irrespective of bactericidal action.

It is impossible to state the relative importance of these different factors in alleviating and curing respiratory infections. I still believe that the sterilizing effect of the chlorine is quite important; but it is possible to discount this factor completely and yet have a rational explanation for the clinical improvement which is undoubted.

The concentration of chlorine that will give the best results should be considered. All of our treatments have been carried out at concentrations varying from 0.013 to 0.015 mgm. per liter. Experience has shown that anything higher than 0.015 mgm. per liter is apt to be irritating and may produce some cough even in normal individuals. It has also been found that when the operator remains in the gassing chamber for two or three hours even at the optimum concentration for a treatment of one hour, he usually develops some slight cough which may last for a day or two. It is quite evident therefore that the best therapeutic results cannot be obtained with concentrations higher than 0.015 mgm. per liter. On the other hand, it has been found that a concentration of 0.009 mgm. per liter is of practically no therapeutic value, even when the treatment is prolonged for two hours. This means that in order to obtain satisfactory results, the concentration of chlorine must be maintained for the entire hour within very narrow limits, and that if the concentration is not so maintained, failures will result that can only discredit the treatment.

If this treatment is to be made available to the medical profession apparatus must be designed that will enable physicians to maintain this concentration exactly, and automatically. Vedder and Sawyer described a small portable apparatus by means of which gaseous chlorine might be released from small cylinders, through a measuring device. The amount of chlorine necessary to set up the desired concentration in any given number of cubic feet is readily calculated. When this apparatus was described it had already been used on about one hundred cases with good results, and it was thought would prove satisfactory. However, subsequent experience has shown that this type of apparatus is most unsatisfactory. The problem is not to set up a concentration that may be correct initially, *but to maintain such a concentration for one hour.* The rate of absorption of chlorine varies enormously with the character of the walls of the room, the number of persons in it, the amount of moisture in the air and many other factors. A room constructed of beaver board absorbs chlorine so rapidly that there are probably no therapeutic amounts of chlorine in such a room, five minutes after the proper amount of gas has been released. This means that in order to maintain a concentration with this type of apparatus, the physician must remain in the room to constantly release such quantities as may be necessary; and he must gauge such quantities by his nose,



Chlorine Inhalator

which will usually be untrained, and which in any case is quite unreliable as the sense of smell becomes dulled after at most ten or fifteen minutes. Even if all this were not so, the physician will not be able to give three or four hours daily to administering such treatments, and if he did he would suffer from excessive dosage of chlorine himself.

For these reasons it was deemed imperative to develop an entirely different type of apparatus. The Wallace and Tiernan Company of Newark, New Jersey, were of precisely the same opinion. It was this Company who had made for us the portable apparatus first described in the article already referred to, and which has been copied by several other manufacturers, although the Wallace and Tiernan Company, who first made it, have refused to sell it for the reasons given. Instead, they devoted their resources for several months to the development of the type of apparatus shown in the figure on plate 34.

A small electric motor in the box operated from the house current, drives a blower delivering air at the outlet of the equipment at a rate of from 10 to 12 cubic feet per minute, the air flow being metered. The chlorine is generated by the electrolysis of hydrochloric acid, the current being supplied by three dry cells. The rate of chlorine generation is controlled by regulating the flow of current through the electrolyte. This is done by means of a rheostat and the current is indicated on a meter. By turning the rheostat until the needle of the meter is on the proper point, the desired concentration is obtained, and will thereafter be delivered constantly with no change. The patient breathes the gas air mixture by adjusting a funnel shaped bag of muslin or paper to his nose and mouth. This fits very loosely so that the air streams past his face, and rebreathing into the bag is avoided. The practising physician can use this apparatus in his office, or can carry it to bed patients.

A different type of apparatus is being made for use in hospitals, institutions and large establishments, where a room will be desired in which to treat a number of patients at one time. Chlorine may be supplied either by a similar electrolytic cell or from a cylinder, but preferably the latter. The amount of chlorine in the air of the room will be determined continuously by a newly devised mechanical chlorine detector. This can be connected electrically with the chlorine generating apparatus, in such a way that more chlorine will be introduced automatically when the concentration in the room

drops below the desired limit, and will be shut off automatically when the concentration reaches the upper limit.

With such apparatus satisfactory therapeutic results can be obtained. They cannot be obtained with any type of apparatus that does not maintain the proper concentration continuously.

COUNTERINDICATIONS

Hay fever, asthma, pneumonia and tuberculosis cannot be benefited by this treatment, and such patients should be excluded.

Hay fever. Even though some slight improvement resulted from the treatment, as soon as the patient goes out a fresh dose of pollen is received, and the condition is as bad as ever. This can only result in discrediting the treatment by offering a false hope of relief.

Asthma. Probably most cases of asthma are caused by sensitization with some variety of foreign protein, and the only hope of relief is the identification and exclusion of the irritating protein. A few cases of asthma are caused by absorption of bacterial protein, following a chronic bronchitis. There is some hope that the chlorine treatment may afford relief to such cases, but at present there is little evidence to this effect. Such cases will have to be selected very carefully for trial. A number of patients suffering from asthma have insisted on giving the treatment a trial. Such cases have been carefully watched, and in nearly every case the chlorine has aggravated the condition and brought on an asthmatic paroxysm, and they have been unable to remain in the gassing chamber the full hour.

Pneumonia. Chlorine has no penetrating power and cannot be expected to have any effect upon portions of the lung where consolidation has occurred.

Tuberculosis. The treatment has not been used on cases of tuberculosis, and cannot be expected to have any specific effect on tubercle bacilli. On the other hand, many cases of chronic pulmonary tuberculosis suffer from chronic bronchitis due to mixed infection, almost as much as from the tuberculosis. It is possible that such cases might obtain some relief from chlorine treatment. However it should be tried in such cases only after further study of the method, and only then in hospitals where it may be accurately controlled.

Hypersusceptibility. A few patients appear to be hypersusceptible to the effects of chlorine, which is to be expected. When in

any case it is evident that the effect of the treatment is far more irritating than it should be, the patient should not remain for the full hour. It is believed that of the small number of patients who receive no benefit from this treatment, a part are hypersusceptible, and the remainder suffer from sinus infection and the affected parts are inaccessible to the gas.

Age presents no counterindication. We have treated one infant of three months suffering from whooping cough, on four separate days. This infant was greatly improved and made a good recovery within two weeks. We have also had several patients suffering from chronic bronchitis ranging from seventy-five to eighty-five years of age. None of these cases were injured by the treatment, and on the contrary reported that they felt much better, that the cough was much less annoying, and that they enjoyed the best nights sleep that they had had in years. Such improvement is worth securing even though little hope can be afforded of the permanent cure of long standing chronic bronchitis.

PROPHYLACTIC USE

The prophylactic use of chlorine is in the future, after the therapeutic value of the treatment is established and methods and apparatus for applying it are on a firm basis. However there is every reason for believing that chlorine may be used in schools, theaters, and other places where people congregate, for the purpose of preventing respiratory infections. In any institution that has a ventilating system, chlorine can be introduced in such quantities as to set up the proper concentration in the rooms supplied. This could be so used in schools for an hour, several times a week.

But even before this can be done, the respiratory infections may be largely controlled through treatment of the early cases. If when colds, influenza, whooping cough or similar infections of the respiratory tract first appear, the first cases are promptly treated, the infection will fail to spread and will not become epidemic. Prompt treatment will thus greatly reduce the number of cases among troops, schools, colleges, and industrial establishments employing large numbers of men and women.

Mention should also be made of the possibility that chlorine may be used successfully in the treatment of certain respiratory diseases of animals. It has thus been used once successfully at Edgewood Arsenal controlling an epidemic of influenza in the horses at Fort

Hoyle, Md. The horses so treated made a complete recovery in much shorter time than could have been expected, and the epidemic ceased to spread. At the time treatment commenced there were 41 cases scattered throughout 14 different stables. Only 3 new cases developed after the first treatment, and at the end of five days all cases had recovered.

It is hoped that this treatment will also prove valuable in treating distemper in dogs. The few cases we have treated appeared to be greatly improved. Much more work will be required to determine the effectiveness of chlorine treatment on the respiratory infections of animals.

REFERENCES

(1) Vedder and Sawyer: Chlorine as a therapeutic agent in certain respiratory diseases. Jour. Amer. Med. Assoc., March 8, 1924, vol. 82, p. 764.

(2) Dakin and Dunham: Handbook of Antiseptics. The Macmillan Co., New York, 1917.

(3) Loevenhart: Benzoylsuperoxyd, ein neues therapeutisches Agent. Therapeutische Monatshefte, 1905, p. 426.

(4) Loevenhart and Grove: Studies on the pharmacological action of oxidizing substances. Jour. Pharm. and Exper. Therap., vol. 3, 101, 131 (1911).

(5) Arkin, Aaron: On the antiseptic and bactericidal action of sodium salts of iodbenzoic, iodoso-and iodoxybenzoic acids. Jour. Pharm. and Exper. Therap., vol. 3, 145 (1911).

(6) Hektoen, Ludvig: Influence of sodium iodoxybenzoate on the production of antibodies in dogs. Transactions Chicago Pathological Society, vol. 8, February 1, 1911.

(7) Arkin, Aaron: The influence of certain oxidizing agents on phagocytosis. Jour. Infectious Diseases, vol. 11, 427 (1912).

(8) Amberg: Samuel: Weitere Untersuchungen über den Einflus der Jodbenzoesäure-Reihe auf entzündliche Reactionen. Zeit. für die gesamte exper. Medizin., vol. 2, 19 (1913).

(9) Loevenhart, A. S.: Certain aspects of biological oxidation. (Harvey Lecture.) Archives of Internal Medicine, vol. 15, 1059 (1915).

(10) Veterinary Bulletin, Office of the Surgeon General, U. S. A., 1924, vol. XIII, 153.

APPENDIX

Conversion Formulas for Toxicity Data

Toxicity data are given in so many different ways by different investigators that it has been found necessary to compile a number of conversion formulas so that the data may be compared with the results secured by our own workers and other investigators.

British publications usually give their figures in parts per million. German chemists use grams per cubic meter or cubic millimeters per cubic meter. Carbon monoxide is almost universally given in per cent. The Chemical Warfare Service, believing that the military value of a toxic agent depends on its toxicity per unit weight, has adopted the uniform standard of milligrams per liter. Interspersed with these modes of expression are a number of others, not as important, but nevertheless troublesome to convert when unexpectedly met with in the course of reading some journal article.

In all the formulas given in this report, the following symbols are used:

m = milligrams per liter.
M = molecular weight of the compound in the vapor state. In the case of practically all solids and liquids this will be the same as the molecular weight as ordinarily considered.
N = Fraction of the molecular weight (number of moles).
P = Atmospheric pressure in millimeters of mercury.
T = Temperature on the absolute scale (Centigrade temperature + 273).
X = Datum in any other system of toxicity figures.

CONVERSION OF DATA TO MILLIGRAMS PER LITER

1. Parts per thousand million

Cubic millimeters per cubic meter (X) $m = \frac{X\,M\,P}{62360000\,T}$

at 25° and 760 mm. $m = \frac{X\,M}{24450000}$

at 0° and 760 mm. $m = \frac{X\,M}{22400000}$

2. Parts per million

Cubic centimeters per cubic meter (X) $m = \frac{X\,M\,P}{62360\,T}$

at 25° and 760 mm. $m = \frac{X\,M}{24450}$

at 0° and 760 mm. $m = \frac{X\,M}{22400}$

3. Parts per thousand (X)

Cubic centimeters per liter $m = \frac{X\,M\,P}{62.36\,T}$

at 25° and 760 mm. $m = \frac{X\,M}{24.45}$

at 0° and 760 mm. $m = \frac{X\,M}{22.4}$

4. Per cent

Parts per hundred (X) $m = \frac{X\,M\,P}{6.236\,T}$

at 25° and 760 mm. $m = \frac{X\,M}{2.445}$

at 0° and 760 mm. $m = \frac{X\,M}{2.24}$

5. Grams per cubic meter (X) $m = X$

6. Milligrams per cubic foot (X) $m = 0.0353\,X$

7. Ounces per 1000 cubic feet (X) $m = X$

8. Ounces per cubic yard (X) $m = \frac{X}{0.027}$

CONVERSION OF MILLIGRAMS PER LITER TO OTHER FORMS

1. Parts per thousand million (X)

Cubic millimeters per cubic meter $X = \frac{62360000\,m\,T}{M\,P}$

at 25° and 760 mm. $X = \frac{24450000\,m}{M}$

at 0° and 760 mm. $X = \frac{22400000\,m}{M}$

2. Parts per million

Cubic centimeter per cubic meter $X = \frac{62360\,m\,T}{M\,P}$

at 25° and 760 mm. $X = \frac{24450\,m}{M}$

at 0° and 760 mm. $X = \frac{22400\,m}{M}$

3. Parts per thousand

Cubic centimeters per liter	$X = \frac{62.36\ m\ T}{M\ P}$
at 25° and 760 mm.	$X = \frac{24.45\ m}{M}$
at 0° and 760 mm.	$X = \frac{22.4\ m}{M}$

4. Per cent

Parts per hundred	$X = \frac{6.236\ m\ T}{M\ P}$
at 25° and 760 mm.	$X = \frac{2.445\ m}{M}$
at 0° and 760 mm.	$X = \frac{2.24\ m}{M}$
5. Grams per cubic meter	$X = m$
6. Milligrams per cubic foot	$X = 28.32\ m$
7. Ounces per 1000 cubic feet	$X = m$
8. Ounces per cubic yard	$X = 0.027\ m$

For the conversion of molecular comparisons expressed in odd numbers, such as 1 part in 2500, 1 part in 8000, etc., a slightly different formula is required.

Let Z be the concentration of the diluent for each part of toxic agent. Any concentration, such as 1 to 2500, 1 to 8000, etc., would be represented by 1 to Z.

Parts per million (X)	$X = \frac{1000000}{Z}$
Milligrams per liter	$m = \frac{M\ P}{0.06236\ Z\ T}$
at 25° and 760 mm.	$m = \frac{M}{0.02445\ Z}$
at 0° and 760 mm.	$m = \frac{M}{0.0224\ Z}$

CHANGE OF VOLUME DUE TO CHANGES IN TEMPERATURE OR PRESSURE

Let V equal the original volume which will be changed to volume V_1 under the influence of a change of temperature from T to T_1 and a change of pressure from P to P_1. Then

Example:

$$V_1 = V \times \frac{P}{P_1} \times \frac{T_1}{T}$$

To convert a concentration of 300 cubic centimeters per cubic meter at 25° and 740 mm. to 0° and 760 mm.

$$V_1 = 300 \frac{740}{760} \times \frac{273}{298} = 268$$

For conversion of any volume to 0° and 760 mm. the equation may be simplified to

$$V_1 = \frac{V\ P}{2.78\ T}$$

Where V is original volume at temperature T and pressure P.

DERIVATION AND USE OF FORMULAS

The formulas given in the appendix may be divided into two main groups:

1. *a.* Molecular concentration of toxic agent compared to total molecular concentration of diluent and toxic agent.
   b. Unit volume of toxic agent per unit volume of diluent.
2. Unit weight of toxic agent per unit volume of diluent.

Under (1) above may be grouped the first four formulas.

1. Parts per thousand million
   Cubic millimeters per cubic meter
2. Parts per million
   Cubic centimeters per cubic meter.
3. Parts per thousand
   Cubic centimeters per liter
4. Parts per hundred
   Per cent

Under (2) may be grouped the last four formulas.

5. Grams per cubic meter
6. Milligrams per cubic foot
7. Ounces per 1000 cubic feet
8. Ounces per cubic yard.

MOLECULAR CONCENTRATION COMPARISONS AND UNIT VOLUME OF TOXIC AGENT PER UNIT VOLUME OF DILUENT

Although the bases for parts per million, parts per thousand, etc., is molecular concentration, the determination of the molecular

concentrations rests on volume comparisons, so that while the two modes of expressing toxicity are theoretically different, the same formulas are used in each case.

Both temperature and pressure are necessary factors with these formulas, for any change in either temperature or pressure will alter the volume of the gaseous toxic agent and thereby change the number of unit volumes in a certain definite space. For example if we volatilize a sufficient amount of a compound to give a concentration of 200 cubic millimeters per cubic meter at 0° temperature and 760 mm. pressure, and later repeat the operation at 25°C. and 760 mm., the concentration in cubic millimeters per cubic meter will be

$$200 \times \frac{298}{273} = 218.3$$

an increase of 9.1 per cent for the same weight of compound. Derivation of the type formula is as follows:

$PV = NRT$ for unit volume comparisons

$\frac{M}{1000\,M} = N$ for 1 cubic meter

$V = X$ where X equals cubic centimeters per cubic meter

$PX = \frac{m}{M} RT$

$m = \frac{P\,X\,M}{R\,T}$ where P is expressed in atmosphere

$R\,T = 22400$

$m = \frac{X\,M}{22400} \times \frac{P}{760} \times \frac{273}{T}$ for any temperature and pressure

$m = \frac{P\,X\,M}{62360\,T}$

Derivation of the type formula for molecular concentrations is as follows:

Let N be the molecular concentration of the toxic gas, and N_L the total molecular concentration per liter. N will depend only on the amount of substance vaporized. N_L will vary according to the temperature and pressure.

$$N : N_L :: X : 1000000$$

$$N = \frac{m}{1000\ M}$$

$$PV = N_L RT \qquad N_L = \frac{PV}{RT} = \frac{P\ 1000}{RT}$$

$$N_L = \frac{1000}{22400} \times \frac{P}{760} \times \frac{273}{T}$$

$$N_L = \frac{P}{62.36\ T}$$

then

$$\frac{m}{1000\ M} : \frac{P}{62.36\ T} :: X : 1000000$$

or

$$\frac{\frac{m}{1000\ M}}{\frac{P}{62.36\ T}} = \frac{X}{1000000}$$

Simplifying:

$$x = \frac{62360\ m\ T}{M\ P}$$

and

$$m = \frac{P\ X\ M}{62360\ T}$$

Some examples are given to show the method of using the formulas.

To convert phosgene datum of 136 parts per million to milligrams per liter at 0° c and 760 mm.

$$m = \frac{X\ M}{22400} \qquad M \text{ for phosgene} = 99$$

$$m = \frac{136 \times 99}{22400} = 0.6$$

To convert chlorine datum of 0.92 milligrams per liter at 27° and 775 mm. to parts per million

$$X = \frac{62360\ m\ T}{M\ P} \qquad M \text{ for chlorine} = 71$$

$$X = \frac{62360 \times 0.92 \times 300}{71 \times 775} = 313$$

To convert phosgene datum of 300 cubic millimeters per cubic meter at standard conditions to milligrams per liter

$$m = \frac{X\ M}{22400000}$$

$$m = \frac{300 \times 99}{22400000} = 0.0013$$

To convert 0.1 parts per thousand of chlorine to milligram per liter at 0° and 760 mm.

$$m = \frac{X\ M\ P}{T\ 62.36}$$

$$m = \frac{0.1 \times 70.92 \times 760}{273 \times 62.36} = 0.32$$

UNIT WEIGHT OF TOXIC AGENT PER UNIT VOLUME OF DILUENT

This type of formula embodies only the simple conversion of the units used. Since the measurements are of unit weight per unit volume in each case, no questions of temperature, pressure, or molecular weight are involved.

For example, the changing of milligrams per cubic foot to milligrams per liter necessitates only the knowledge of the number of liters in a cubic foot, which is *28.32* A concentration of 100 milligrams per cubic foot $= \frac{100}{28.32} = 3.53$ milligrams per liter.

In the same way the conversion of milligrams per liter to ounces per 1000 cubic feet is secured. 1 ounce = 28,350 milligrams. 1000 cubic feet = 28,317 liters.

Therefore to change milligrams per liter to ounces per 1000 cubic feet it is necessary to divide by 28,350 and multiply by 28,317

$$\frac{m\ 28{,}317}{28{,}350} = X$$

$$X = m \times 0.9989$$

For practical purposes $X = m$

and a concentration of 0.6 milligrams per liter is the same as 0.6 ounces per 1000 cubic feet.

Table of atomic weights

NAME	SYMBOL	ATOMIC WEIGHT	NAME	SYMBOL	ATOMIC WEIGHT
Aluminium	Al	27.0	Molybdenum	Mo	96.0
Antimony	Sb	120.2	Neodymium	Nd	144.3
Argon	A	39.9	Neon	Ne	20.2
Arsenic	As	74.96	Nickel	Ni	58.68
Barium	Ba	137.37	Niton	Nt	222.4
Bismuth	Bi	209.0	Nitrogen	N	14.008
Boron	B	10.9	Osmium	Os	190.9
Bromine	Br	79.92	Oxygen	O	16.00
Cadmium	Cd	112.40	Palladium	Pd	106.7
Calcium	Ca	40.07	Phosphorus	P	31.04
Carbon	C	12.005	Platinum	Pt	195.2
Cerium	Ce	140.25	Potassium	K	39.10
Cesium	Cs	132.81	Praseodymium	Pr	140.9
Chlorine	Cl	35.46	Radium	Ra	226.0
Chromium	Cr	52.0	Rhodium	Rh	102.9
Cobalt	Co	58.97	Rubidium	Rb	85.45
Columbium	Cb	93.1	Ruthenium	Ru	101.7
Copper	Cu	63.57	Samarium	Sa	150.4
Dysprosium	Dy	162.5	Scandium	Sc	45.1
Erbium	Er	167.7	Selenium	Se	79.2
Europium	Eu	152.0	Silicon	Si	28.1
Fluorine	F	19.0	Silver	Ag	107.88
Gadolinium	Gd	157.3	Sodium	Na	23.00
Gallium	Ga	70.1	Strontium	Sr	87.63
Germanium	Ge	72.5	Sulphur	S	32.06
Glucinum	Gl	9 1	Tantalum	Ta	181.5
Gold	Au	197.2	Tellurium	Te	127.5
Helium	He	4.00	Terbium	Tb	159.2
Holmium	Ho	163.5	Thallium	Tl	204.0
Hydrogen	H	1.008	Thorium	Th	232.15
Indium	In	114.8	Thulium	Tm	169.9
Iodine	I	126.92	Tin	Sn	118.7
Iridium	Ir	193.1	Titanium	Ti	48.1
Iron	Fe	55.84	Tungsten	W	184.0
Krypton	Kr	82.92	Uranium	U	238.2
Lanthanum	La	139.0	Vanadium	V	51.0
Lead	Pb	207.20	Xenon	Xe	130.2
Lithium	Li	6.94	Ytterbium (neoytterbium)	Yb	173.5
Lutecium	Lu	175.0			
Magnesium	Mg	24.32	Yttrium	Yt	89.33
Manganese	Mn	54.93	Zinc	Zn	65.37
Mercury	Hg	200.6	Zirconium	Zr	90.6

Molecular weights of some of the more common toxic cases

NAME	FORMULA	MOLECULAR WEIGHT
Arsenic trichloride	$AsCl_3$	181.34
Arsine	AsH_3	77.98
Benzyl bromide	$C_6H_5CH_2Br$	171.01
Brombenzyl cyanide	$C_6H_5CHBrCN$	196.02
Capsaicin		305.0
Carbon monoxide	CO	28.0
Chloracetophenone	$C_6H_5COCH_2Cl$	154.56
Chlorine	Cl_2	70.92
Chlorpicrin	Cl_3CNO_2	164.38
Cyanogen chloride	CNCl	61.47
Cyan D.A.	$(C_6H_5)_2AsCN$	254.96
Cyan D.M.	$NH(C_6H_4)_2AsCN$	267.96
D.A. (diphenyl chlor arsine)	$(C_6H_5)_2AsCl$	264.42
D.M. (diphenyl amine chlorarsine)	$NH(C_6H_4)_2AsCl$	277.42
D.A. oxide	$(C_6H_5)_2As)_2O$	474.08
D.M. oxide	$(NH(C_6H_4)_2As)_2O$	500.06
Ethyl arsenious oxide	C_2H_5AsO	120.0
Ethyl dichlor arsine	$C_2H_5AsCl_2$	174.92
Hydrogen cyanide	HCN	27.0
Hydrogen sulphide	H_2S	34.08
M-1	$CHCl{:}CHAsCl_2$	207.34
Methyl arsenious oxide	CH_3AsO	105.98
Methyl dichlorarsine	CH_3AsCl_2	160.9
Mustard	$(ClCH_2CH_2)_2S$	159.04
Mustard sulphone	$(ClCH_2CH_2)_2SO_2$	191.04
Mustard sulphoxide	$(ClCH_2CH_2)_2SO$	175.04
Palite	$ClCO_2CH_2Cl$	128.94
Phosgene	$COCl_2$	98.92
Superpalite	Cl_3CCOCl	181.84

Conversion table for gases: Parts per million versus milligrams per liter

(25°C. and 760 mm. mercury, barometric pressure)

MOLECULAR WEIGHT	1 MGM./L.= P.P.M.	1 P.P.M.= MGM./L.	MOLECULAR WEIGHT	1 MGM./L.= P.P.M.	1 P.P.M.= MGM./L.	MOLECULAR WEIGHT	1 MGM./L.= P.P.M.	1 P.P.M.= MGM./L.	MOLECULAR WEIGHT	1 MGM./L.= P.P.M.	1 P.P.M.= MGM./L.	MOLECULAR WEIGHT	1 MGM./L.= P.P.M.	1 P.P.M.= MGM./L.	MOLECULAR WEIGHT	1 MGM./L.= P.P.M.	1 P.P.M.= MGM./L.
1	24,450	0.0000409	51	479	0.002086	101	242.1	0.00413	151	161.9	0.00618	201	121.6	0.00822	251	97.4	0.01027
2	12,230	0.0000818	52	470	0.002127	102	239.7	0.00417	152	160.9	0.00622	202	121.0	0.00826	252	97.0	0.01031
3	8,150	0.0001227	53	461	0.002168	103	237.4	0.00421	153	159.8	0.00626	203	120.4	0.00830	253	96.6	0.01035
4	6,113	0.0001636	54	453	0.002209	104	235.1	0.00425	154	158.8	0.00630	204	119.9	0.00834	254	96.3	0.01039
5	4,890	0.0002045	55	445	0.002250	105	232.9	0.00429	155	157.7	0.00634	205	119.3	0.00838	255	95.9	0.01043
6	4,075	0.0002454	56	437	0.002290	106	230.7	0.00434	156	156.7	0.00638	206	118.7	0.00843	256	95.5	0.01047
7	3,493	0.0002863	57	429	0.002331	107	228.5	0.00438	157	155.7	0.00642	207	118.1	0.00847	257	95.1	0.01051
8	3,056	0.000327	58	422	0.002372	108	226.4	0.00442	158	154.7	0.00646	208	117.5	0.00851	258	94.8	0.01055
9	2,717	0.000368	59	414	0.002413	109	224.3	0.00446	159	153.7	0.00650	209	117.0	0.00855	259	94.4	0.01059
10	2,445	0.000409	60	408	0.002554	110	222.3	0.00450	160	152.8	0.00654	210	116.4	0.00859	260	94.0	0.01063
11	2,223	0.000450	61	401	0.002495	111	220.3	0.00454	161	151.9	0.00658	211	115.9	0.00863	261	93.7	0.01067
12	2,038	0.000491	62	394	0.00254	112	218.3	0.00458	162	150.9	0.00663	212	115.3	0.00867	262	93.3	0.01072
13	1,881	0.000532	63	388	0.00258	113	216.4	0.00462	163	150.0	0.00667	213	114.8	0.00871	263	93.0	0 01076
14	1,746	0.000573	64	382	0.00262	114	214.5	0.00466	164	149.1	0.00671	214	114.3	0 00875	264	92.6	0.01080
15	1,630	0.000614	65	376	0.00266	115	212.6	0.00470	165	148.2	0.00675	215	113.7	0.00879	265	92.3	0.01084
16	1,528	0.000654	66	370	0.00270	116	210.8	0.00474	166	147.3	0.00679	216	113.2	0.00883	266	91.9	0.01088
17	1,438	0.000695	67	365	0.00274	117	209.0	0.00479	167	146.4	0.00683	217	112.7	0.00888	267	91.6	0.01092
18	1,358	0.000736	68	360	0.00278	118	207.2	0.00483	168	145.5	0.00687	218	112.2	0.00892	268	91.2	0.01096
19	1,287	0.000777	69	354	0.00282	119	205.5	0.00487	169	144.7	0.00691	219	111.6	0.00896	269	90.9	0.01100
20	1,223	0.000818	70	349	0.00286	120	203.8	0.00491	170	143.8	0.00695	220	111.1	0.00900	270	90.6	0.01104
21	1,164	0.000859	71	344	0.00290	121	202.1	0.00495	171	143.0	0.00699	221	110.6	0.00904	271	90.2	0.01108
22	1,111	0.000900	72	340	0.00294	122	200.4	0.00499	172	142.2	0.00703	222	110.1	0.00908	272	89.9	0.01112
23	1,063	0.000941	73	335	0.00299	123	198.8	0.00503	173	141.3	0.00708	223	109.6	0.00912	273	89.6	0.01117
24	1,019	0.000982	74	330	0.00303	124	197.2	0.00507	174	140.5	0.00712	224	109.2	0.00916	274	89.2	0.01121

25	978	0.001022	75	326	0.00307	125	195.6	0.00511	175	139.7	0.00716	225	108.7	0.00920	275	88.9	0.01125
26	940	0.001063	76	322	0.00311	126	194.0	0.00515	176	138.9	0.00720	226	108.2	0.00924	276	88.6	0.01129
27	906	0.001104	77	318	0.00315	127	192.5	0.00519	177	138.1	0.00724	227	107.7	0.00928	277	88.3	0.01133
28	873	0.001145	78	313	0.00319	128	191.0	0.00524	178	137.4	0.00728	228	107.2	0.00933	278	87.9	0.01137
29	843	0.001186	79	309	0.00323	129	189.5	0.00528	179	136.6	0.00732	229	106.8	0.00937	279	87.6	0.01141
30	815	0.001227	80	306	0.00327	130	188.1	0.00532	180	135.8	0.00736	230	106.3	0.00941	280	87.3	0.01145
31	789	0.001268	81	302	0.00331	131	186.6	0.00536	181	135.1	0.00740	231	105.8	0.00945	281	87.0	0.01149
32	764	0.001309	82	298	0.00335	132	185.2	0.00540	182	134.3	0.00744	232	105.4	0.00949	282	86.7	0.01153
33	741	0.001350	83	295	0.00339	133	183.8	0.00544	183	133.6	0.00748	233	104.9	0.00953	283	86.4	0.01157
34	719	0.001391	84	291	0.00344	134	182.5	0.00548	184	132.9	0.00753	234	104.5	0.00957	284	86.1	0.01162
35	699	0.001432	85	288	0.00348	135	181.1	0.00552	185	132.2	0.00757	235	104.0	0.00961	285	85.8	0.01166
36	679	0.001472	86	284	0.00352	136	179.8	0.00556	186	131.5	0.00761	236	103.6	0.00965	286	85.5	0.01170
37	661	0.001513	87	281	0.00356	137	178.5	0.00560	187	130.7	0.00765	237	103.2	0.00969	287	85.2	0.01174
38	643	0.001554	88	278	0.00360	138	177.2	0.00564	188	130.1	0.00769	238	102.7	0.00973	288	84.9	0.01178
39	627	0.001595	89	275	0.00364	139	175.9	0.00569	189	129.4	0.00773	219	102.3	0.00978	289	84.6	0.01182
40	611	0.001636	90	272	0.00368	140	174.6	0.00573	190	128.7	0.00777	240	101.9	0.00982	290	84.3	0.01186
41	596	0.001677	91	269	0.00372	141	173.4	0.00577	191	128.0	0.00781	241	101.5	0.00986	291	84.0	0.01190
42	582	0.001718	92	266	0.00376	142	172.2	0.00581	192	127.3	0.00785	242	101.0	0.00990	292	83.7	0.01194
43	569	0.001759	93	263	0.00380	143	171.0	0.00585	193	126.7	0.00789	243	100.6	0.00994	293	83.4	0.01198
44	556	0.001800	94	260	0.00384	144	169.8	0.00589	194	126.0	0.00793	244	100.2	0.00998	294	83.2	0.01202
45	543	0.001840	95	257	0.00389	145	168.6	0.00593	195	125.4	0.00798	245	99.8	0.01002	295	82.9	0.01207
46	532	0.001881	96	255	0.00393	146	167.5	0.00597	196	124.7	0.00802	246	99.4	0.01006	296	82.6	0.01211
47	520	0.001922	97	252	0.00397	147	166.3	0.00601	197	124.1	0.00806	247	99.0	0.01010	297	82.3	0.01215
48	509	0.001963	98	249.5	0.00401	148	165.2	0.00605	198	123.5	0.00810	248	98.6	0.01014	298	82.0	0.01219
49	499	0.002004	99	247.0	0.00405	149	164.1	0.00609	199	122.9	0.00814	249	98.2	0.01018	299	81.8	0.01223
50	489	0.002045	100	244.5	0.00409	150	163.0	0.00613	200	122.3	0.00818	250	97.8	0.01022	300	81.5	0.01227

Factors for conversion of some units

1. Mgm. per L. times 28.32 = Mgm. per cu. foot.
2. Mgm. per L. times 1,000 = Mgm. per cu. meter.
3. Mgm per cu. foot times 35.314 = Mgm. per cu. meter.
4. Mgm. per cu. meter times 0.02832 = Mgm. per cu. foot.

AUTHOR INDEX

Achard, 251.
Adams, 175.
Amberg, S., 299, 304.
Arkin, Aaron, 304.

Barcroft, 123.
Barcroft, Hunt and Dufton, 120, 122.
Barry, 114, 123.
Beebe, 265, 292.
Belli, 291.
Berghoff, 253.
Brooke, I. E., 293.
Brown, Carl H., preface.
Bunsen, 13.

Cushny, 282, 293.

Dakin and Dunham, 297, 304.
Diodorus Siculus, 292.
Douglas, 111, 123.
Dudley, S. F., 293.
Dunn, 92, 122, 123.

Edkins and Tweedy, 92, 123.
Elliott, T. R., 248.

Fairlie, 286, 293.
Fieldner, Katz and Kinney, 286, 293.
Fischer, 97.
Flury, 181, 183.
Flury and Wieland, 165.
Francine, Dr. Albert P., 255–258.
Fries, General Amos A., 9.
Fries and West, 200, 214, 215, 269, 292.

Gilchrist, 122, 255.
Groot, 264.

Haber, Professor, 2.
Haggard and Henderson, 123, 195.
Hains, Paul, 292.
Haldane, 105, 117, 121, 191, 196, 227, 239.
Hawes, J. B., 248.
Hektoen, L., 299, 304.
Hill, 122.
Hill and Lupton, 111, 123.
Hine, Lt. Col., 292.
Hoover, C. R., 289, 293.
Hunt and Jones, 122, 123.

Irvine, 293.

Jones, Ernest, 269, 292.

Karsner, 123.
Klotz, 122.
Krogh, 96.

LaCoste, 173, 175.
Laqueur and Magnus, 92, 113, 114, 123.
Lefebure, xiii, 183.
Lehmann, K. B., 281, 293.
Lewis, Professor, 157.
Loevenhart, A. S., 299, 304.
Loevenhart and Grove, 304.
Ludendorff, 174.
Lynch, Smith and Marshall, 165.

Magnus, 281, 293.
Mahan, Admiral, 1.
Manning, Mr. V. H., 6.
Marshall, Lynch and Smith, 165.
Mayer, 100.
McCutcheon, 265, 292.
Meakins and Priestly, 249.
Meek and Eyster, 123.
Michaelis, 175.
Milham, Willis I., 61.
Monroe, Randolph, 212.

O'Ryan, Major General, 255.

Paris, Matthew, 265.
Peters, R. A., Captain, 248.

Sandall, 246.
Schaeffer, 191.
Schneider and Truesdell, 100, 123.
Schwarte, 265, 292.
Shufflebotham, 167, 172.
Sihle, 281, 293.
Smith, Lorraine, 123.
Sollman, 184, 195.
Stewart, 102.
Stewart, Matthew J., 145, 166.
Symons, 285, 293.

Thucidides, 292.

Underhill, 111, 123.

Vedder and Sawyer, 304.

Walton, Duncan C., preface, 264.
Warthin and Weller, 154–156, 165.
Williams, 166.
Wilson and Mackintosh, 147, 166, 251.
Winternitz, 166.

SUBJECT INDEX

Acids, fatty, 30.
Acyl halides, 31.
Adamsite, 175.
Adiabatic changes, 43.
Adsorption, 12.
Aid stations, special supplies for, 236.
Air-craft as chemical warfare distributors, 269.
Alarm, gas, 219.
Alcohols, 26.
Aldehydes, 28.
Alkyl halides, 25.
Ambrine, use of for mustard burns, 155.
Ambulance stations, 239.
American attitude in gas at Hague Conference, 1.
gas casualties, XII.
Amines, 33.
Ammonia canister supplied for protection, 282.
inhalations after phosgene poisoning, 113.
on ships, 281.
Anhydrides, 31.
Anoxaemia, blue and grey types, 105.
effect of, on pulse, 100.
effect of, on the respiration, 106.
results of, in phosgene poisoning, 103.
types of, 104.
Armanent, limitation of, XIII.
Aromatic hydrocarbons, nomenclature of, 36.
Arsenic in organs following poisoning by irritant smokes, 177.
Arsenical poisoning following lewisite burns, 163.
Arsine, 34, 173.
in submarines, 290.
physiologic action of, 192.
properties of, 191.
protection against, 192.
Atmosphere, adiabatic changes in, 43.
composition of, 40.
heating and cooling of, 42.
dust in, 41.
moisture in, 49.
nocturnal stability of, 44.
pressure of, 41.
Atmospheric temperature, 45.
Automobiles, production of carbon monoxide by, 190.
Avogadro's law, 17.

Bactericidal effect of chlorine, 294.
Bathing after exposure to mustard, 151.
units, 226.
system used, 228.
Battle dressing stations, supplies for, 277.
Benzyl alcohol, use of, to relieve itching, 157.
Black veil respirator, 197.
Blister fluid, is mustard in?, 143.
Blood changes in phosgene poisoning, 98, 99.
count after mustard poisoning, 145.
pressure after phosgene poisoning, 101.
Blue cross shell, 73, 129, 173, 174.
Boots for horses, 215.
Boyle's law, 15.
Bromacetone, 167.
Brombenzylcyanide, properties of, 168.
removal of, 169.
Bronchial spasm in chlorine poisoning, 88.
Broncho-pneumonia after mustard poisoning, 144, 155.
Bureau of mines, 6, 7.
Burns from lewisite, 163.

Caffeine, use of, in pulmonary oedema, 111, 117.

Calculation of concentrations, 19, appendix.
Canaries, use of, to warn of presence of carbon monoxide, 191.
Canister, does not protect against all gases, 207.
protection afforded against formaldehyde, 285.
protection afforded against hydrocyanic acid, 284.
protection afforded against nitrous fumes, 286.
protection afforded against Sulphur dioxide, 287.
theoretical requirements for, 205.
various types of, 206.
Carbon dioxide in blood in pulmonary oedema, 105.
mixed with oxygen, 117.
Carbon monoxide, 76.
chemical detector for, 289.
physiological action of, 188.
poisoning, pathology of, 190.
poisoning, after effects, 191.
produced by automobiles, 190.
production of, by explosives, 188.
symptoms of poisoning by, 189.
Casualties caused by Lachrymators, 167.
caused by mustard, 129.
gas, organization to care for, 233.
Cell penetration by mustard, 132, 134, 138.
Charcoal, 12.
for gas mask, 201, 203.
Charles Law, 16.
Chemical industry of Germany, XIII.
Warfare Service, development of, 6, 10.
Chloracetophenone, burning of skin by, 171.
properties of, 170.
removal of, 170.
toxicity of, on dogs, 171.
use against mobs, 172.
Chloride of lime for removing mustard, 152, 223, 224.
Chlorine, action of in stimulating response of body to infection, 298.
action of, on D.A., 175.
action on mustard, 126.
characteristics of, 77.
concentration required for treatment, 300.
counterindications to use of, 302.
cylinders, 77.
delayed death from, 84.
disability caused by, 249.
immediate death from, 84.
in treatment of respiratory diseases, 294.
poisoning, pathology of, 85, 87.
prophylactic use of, 303.
physiological action of, 88.
symptoms of poisoning, 84.
treatment, results of, 295.
use of, to destroy mustard on clothing, 226.
used to control horse influenza, 303.
Chlorpicrin, 25.
characteristics of, 81.
persistency of, 68.
poisoning, pathology of, 109.
removal of, 83.
symptoms produced by, 108.
Classification of chemical warfare agents, 69.
Clothing, protective, 213.
removal of gas from, 225.
to be carried by bathing unit, 227.
Cloud gas, 3.
detection of, 219.
when used, 217.
Clouds, formation of, 53.
types, 52.
Coefficient of expansion as applied to shell, 15.
Collecting stations, location and function, 236.
supplies for, 239.
Concentration, calculation of, 19, appendix.
of chlorine required in treatment of respiratory infections, 300.

Conference on limitation of armament, XIII.
Convalescent hospitals, 157, 242.
Convalescents, management of, 157.
Corrosion of chlorine cylinders, 77.
Counterindications to use of chlorine, 302.
Cyanogen chloride, 283.
Cyclones, extratropical, 55, 57.
tropical, 55.

Dalton's law, 18.
Dakin's solution, use of, in treatment of mustard burns, 156.
Decomposing organic matter produces lack of oxygen in inclosed spaces, 291.
Degassing ships, 273.
Development of Chemical Warfare Service, 6.
Dewpoint, 51.
Diagnosis of gassing by irritant smokes, 182.
of pulmonary irritant poisoning, 110.
Dichloramine T, use of in treatment of mustard burns, 154, 156.
Dichlorethylsulphide, *see* Mustard.
Diffusion, 11, 18.
of smokes, 62.
Digitalis, use of, in pulmonary oedema, 117.
Diphenylaminechlorarsine, properties of, 175.
Diphenylchlorarsine, introduction of, 5.
properties of, 173, 175.
Diphenylcyanarsine, 5, 173.
Diphosgene, 81.
Disability caused by gas, XI, 245–255.
Disinfection of gas masks, 210.
Disposition of cases gassed by irritant smokes, 181.
of gassed cases on shipboard, 278.
Drill, gas mask, 207.
Dugouts, carbon monoxide in, 188.
Duty, test to determine fitness for, 119.

Edgewood Arsenal, origin of, 8.
Arsenal, capacity of, 9.
Efficiency of gas warfare, XI.
Effort syndrome following mustard poisoning, 148.
Emetine, use of, in pulmonary oedema, 114.
Emphysema following chlorine poisoning, 87, 88.
Esters, 31.
Ethers, 33.
Ethyldichlorarsine, 173.
Eye injuries after mustard, 141, 148.

Ferric hydroxide and adsorption, 13.
paste, preparation of, 165.
Filters, gas mask for irritant smokes, 201, 205.
Fire extinguisher fumes, 286.
First aid after gassing by pulmonary irritants, 110.
aid for mustard poisoning, 151.
Flammenwerfers, 265.
Fogs, 51.
Foretelling use of gas, 217.
Formaldehyde, 284.
Frost, prediction of, 60.

Gas alarms, 219.
attack, the first, 2.
attacks, conditions favorable to, 67.
attacks, Indications of impending, 219.
attacks, Malingering after, 110.
British output, 9.
casualties, American, XII.
casualties, organization to care for, 233.
casualties, ratio to wounded, 243.
cylinders, objections to, 3.
cylinders, portable, 18.
defense, organization of, 216.
defense, instructions for, 230.
defense service, 7–8.
foretelling use of, 217.
German output, 9.
mask, description of present type, 200.
mask, disinfection of, 210.

Gas mask, does not protect against all gases, 207.
mask drill, 207.
mask, development of, 196.
mask, extemporized, 270.
masks, the first, 2.
Masks, first American, 6.
mask filters, 201, 205.
masks, French, development of, 199.
mask, German, 199.
mask for head injuries, 212, 275.
mask for horses, 214.
mask, inspection of, 210.
masks, Naval types described, 270.
masks, proportion of various sizes of, 202.
masks, requirements of, 196, 205.
officers, 216.
proof shelters, 221.
proof shelters, disadvantages of, 224.
proof shoes and gloves, 271.
protection of supplies from, 229.
removal from clothing, 225.
removal from surroundings, 224.
shell, first use of, 3.
sentries, duties of, 220.
service, A. E. F., 9.
troops, number required, XIII.
warfare, disability caused by, XI, 244.
warfare, efficiency of, XI.
warfare, is it humane?, X.
warfare, prejudice against, IX.
Gassed animals, rate of recovery in, 259.
cases American, examination of, 254.
cases on ships, disposition of, 278.
cases, rules for transportation of, 241.
cases, tuberculosis in, 248, 251, 252–254, 256–258.
cases, ultimate disposition of, 244.
Gases, American production of, 1, 9.
effect of terrain and weather on, 66.
list of, 70.
persistency of, 68.
tactical use of, 68.
Gasoline fumes, 290.
German Chemical industry, XIII.
Graham's Law, 18.
Gram molecule, 17.
Greek fire, 1, 264.
Green cross shell, 73, 129, 174.
Gum glucose intravenous therapy, 114.
Gutzeit test for water contaminated for arsenical compounds, 183.

Hague Congress, American attitude on gas, 1.
Hail, 54.
Head injuries, gas mask for, 212, 275.
Heart in phosgene poisoning, 102, 103.
Hemoptysis after gassing, 251.
Hering and Breuer reflex, 121.
Horse influenza, controlled by chlorine, 303.
Horses, protection for, 214.
resistant to chloracetophenone, 171.
Hospital center, 240.
ships, liable to be gassed, 280.
Hospitalization after pulmonary irritants, 119.
after mustard, 251.
Humidity, absolute and relative, 50.
effect on gas, 67.
Hydrocarbons, 21.
aromatic, 35.
aliphatic, 21.
unsaturated, 24.
Hydrocyanic acid, 19, 76, 283.
acid, delayed death following poisoning by, 187.
acid, lethal dose of, 186.
acid, physiologic action of, 184.
acid, symptoms of poisoning by, 186.
Hydrogen sulphide on ships, 288.
Hydrolysis of mustard, 126, 131, 132.
Hypo helmet, 197.

Illuminating gas, danger of, 190.
Incendiaries, 194.

Infection after pulmonary oedema, prevention of, 118.
Inhalator, portable for chlorine treatment, 301.
Insolation, effects of, 42–44.
Inspection of gas masks, 210–212.
Instruction for gas defense, 230.
Instruments, protection from gas, 229.
Intoxication general, following mustard poisoning, 139, 145.
Invalidism after mustard poisoning, 149.
after pulmonary irritants, 119.
Irritable heart after gassing, 250, 253.
Irritant gases, 69.
smokes, 173.
smokes, diagnosis of cases affected by, 182.
smokes, first use of, 5.
smokes, irritation points, 179.
smokes, pathology following poisoning by, 177.
smokes, poisoning of water by, 183.
smokes, symptoms in man, 178.
smokes, treatment of casualties produced by, 182.
Isobaric charts, 41.

Kerosene for removing mustard, 152.
Ketones, 28.
Kieselguhr, use of, in canister filling, 204.

Lachrymators, 69, 167.
naval use of, 268.
treatment for, 171.
Lachrymatory shell, 4.
Laryngitis after mustard poisoning, 143.
Latent period in mustard poisoning, 138.
Lethal dose, hydrocyanic acid, 186.
points of lewisite, 158.
Lewisite burns, 163.
characteristics of, 157.
manufacture of, 158.
pathology of, 160.
poisoning, treatment of, 164.
rate of recovery of animals gassed with, 263.
symptoms of poisoning by, 159.
List of chemical warfare agents, 70.
Livens projector, 4, 219.
Lung irritants possible naval use, 267.
weight in phosgene poisoning, 91.
Lung-heart ratios in phosgene poisoning, 93.

Malingering after gas attacks, 110.
Manufacture of chlorine, 77.
Mask, gas, development of, 196.
Matter, structure of, 11.
Medical Department activities, 7–8.
examination of gassed cases, 246.
Research Division, organization of, 10.
Mercaptans, 28.
Microscopic changes in phosgene poisoning, 92.
Mines, carbon monoxide in, 188.
Ministry of Pensions, figures supplied by, 245.
Mobs, control of, by gas, XI, 172.
Moisture in atmosphere, 49.
Mole, definition of, 17.
Monsoons, 48.
Morphine, use counterindicated in pulmonary oedema, 118.
Mortar, stokes, 4.
Mustard, action on ferments, 133.
as a casualty producer, 129.
burns, common locations of, 142.
burns followed by pigmentation, 142.
boots to protect horses, 215.
characteristics of, 125.
casualties, treatment of, 153.
casualties, treatment by bathing units, 226.
disability caused by, 250.
effect of weather on, 13.
eye injuries after, 141, 148.
fate of, in body, 131.
gas, first use of, 4–5.
general intoxication, 139, 145.

Mustard, in blister fluid, 143.
latent period, 138.
lethal dose, 128.
oil of, and mustard gas, 33.
poisoning, pathology of. 134.
pharmacological action of, 129.
poisoning, effect of, on blood count, 145.
poisoning, effort syndrome after, 148.
poisoning, first aid, 151.
poisoning, functional photophobia in, 147, 154.
poisoning, invalidism after, 149.
poisoning, prognosis in, 138, 146.
poisoning, results analyzed, 147.
proof clothing on shipboard, 271.
prophylaxis, 149.
protective clothing for, 213.
rate of recovery of animals after gassing with, 262.
solubility of, 126, 128.
sulphone, 126, 132.
sulphoxide, 126.
symptoms of poisoning, 138.
theories of action, 132.
vapor tension of, 127.

Naval gas defense, 272.
methods of degassing, 273.
methods of releasing gas, 269.
tactics different from Army, 266.
types of gas mask, 270.
Negroes resistant to mustard, 150.
Nervous changes following poisoning by irritant smokes, 180.
Neurasthenia after gassing, 121, 246, 255, 263.
Nitriles, 32.
Nitrous fumes from burning explosives, 192.
fumes, poisoning on shipboard by, 285.
fumes, use of gas mask for, 193.
Nocturnal dyspnoea following pulmonary irritants treated by oxygen in chambers, 120.
stability of atmosphere, 44.

Oedema, Fischers theory of, 97.
Ointments, protective, 150.
Ordnance Department, 8.
Organization of bathing unit, 227.
of gas defense, 216.
Oxygen, administration of, in carbon monoxide poisoning, 191.
administration of, in pulmonary oedema, 106, 115.
chambers, use in nocturnal dyspnoea, 120.
cylinders for Hospital ships, 280.
cylinders on ambulances, 239.
effects of lack of, 291.
inhalators, 6.
not practicable on fighting ships, 277.
pure effect of, on the lungs, 116.
requirement of body, 110–111.
versus venesection in pulmonary oedema, 111.
with carbon dioxide, 117.
Ozone, general properties, 193.

Palite, 80.
Pathology of chlorine poisoning, 85.
of chlorpicrin poisoning, 109.
of mustard poisoning, 134.
of phosgene poisoning, 90–95.
following poisoning by lewisite, 160.
of poisoning by irritant smokes, 176.
Persistency of various agents, 68.
Pigmentation following mustard burns, 142.
Pharmacological action of mustard, 129.
Phenols, 37.
Phosgene, characteristics of, 78–80.
effect of weather on, 67.
first use of, 3.
physiologic action of, 94.
poisoning, acute death from, 94.
poisoning, blood changes in, 98, 99.
poisoning, the circulatory changes in, 100, 102.

Phosgene, poisoning, cause of pulmonary oedema, 95–98.
poisoning, cause of sudden deaths, 104.
poisoning, delayed action, 89.
poisoning, development of pathological changes, 92.
poisoning, inhalation of ammonia after, 113.
poisoning, pathology of, 90–95.
poisoning, pneumonia after, 95.
poisoning, prevention of pneumonia, 118.
poisoning, use of gum glucose solutions in, 114.
poisoning, use of digitalis and strophanthin in, 117.
poisoning, possible use of urease in, 114.
poisoning, types of, 89.
produced from carbon tetrachloride in fire extinguishers, 287.
rate of recovery of animals after gassing, 260.
tests for, 80.
Phosphine, 34, 289.
Phosphorus, 194.
Photophobia, functional, after mustard, 250.
Physiologic action of arsine, 192.
action of carbon monoxide, 188.
action of phosgene, 94.
classification of chemical warfare agents, 69.
Pneumonia after phosgene poisoning, 95, 118.
Polycythaemia after pulmonary irritants, 99, 119.
Prejudice against gas warfare, IX.
Preparedness, necessity for, 2.
Prognosis after gassing by pulmonary irritants, 118.
in mustard poisoning, 138, 146.
Projectors, Livens, 41, 219.
Prophylaxis carbon monoxide poisoning, 191.
for mustard, 149.
nitrous fumes, 193.
Protection for horses, 214.
of supplies from gas, 229.
Protective clothing, 213.
ointments, 150.
Pulse in phosgene poisoning, 100.
Pulmonary circulation in phosgene poisoning, 102.
irritants, 69, 77.
irritants, classification of chronic cases, 122.
irritants, first aid for, 234.
irritants, hospitalization after, 119.
irritants, neurasthenia following, 121.
irritants, polycythaemia following poisoning by, 119.
irritants, relative toxicity of, 83.
oedema, 84.
oedema, anoxaemia in, 104.
oedema, cause of, in phosgene poisoning, 95–98.
oedema, caused by nitrous fumes, 193.
oedema, effects on carbon dioxide content of blood, 105.
oedema, result of chlorine poisoning, 88.
oedema, treatment by intravenous saline, 113.
oedema, use of digitalis in, 117.
oedema, use of emetine in, 114.
oedema, use of gum glucose solution in, 114.
oedema, use of morphine contraindicated, 118.
oedema, venesection in, 112.

Rain, 54.
Ratio of gas casualties to wounded, 243.
Recovery, rate of, in gassed animals, 259.
Reflex of Hering and Breuer, 121.
of Knoll and Franck, 100.
Regimental aid stations, 235.
Removal of gas, 224.
Reports made by gas officers, 231.
Respiration, effects of anoxaemia on, 106.

Results of mustard poisoning analyzed, 147.
Rubber, solubility of mustard in, 126.
Rules, general for transportation of gassed cases, 241, 242.

Saline solution intravenously in pulmonary oedema, 113.
Sanitary Corps, 7.
Senate Committee, testimony of Dr. Francine before, 255.
Sensitivity to mustard, 149, 150.
Sentries gas, duties of, 220.
Shell fillings, 72–75.
  hole water, poisoning by, 183, 229.
  holes, treatment to remove mustard, 225.
Shelters, gas proof, 221.
Shoes, treatment to render gas proof, 271.
Sizes of gas masks, 202.
Skagerrack, battle of, 266.
Smoke, classes of, 63.
  clouds, movements of, 64.
  diffusion of, 62.
  irritant, 173–183.
  screening, 63, 194.
  settling of, 62.
  total obscuring power, 64.
  toxic, 63.
Soda lime in gas mask, 201.
Soldiers heart, 121.
Sputum in phosgene poisoning, 90.
Sternutators, 69.
Stokes, mortar, 4.
Strophanthin, use of, in pulmonary oedema, 117.
Subjective symptoms, relief of, in pulmonary oedema, 117.
Sublimation, 14.
Submarines, possibility of gas attacks on, 279.
Sudden death from phosgene poisoning, 89, 104.
Sulphur dioxide as fumigant, 287.
Superpalite, 81.
Supplies, protection from gas, 229.
Surface tension, 12.
Symptoms following poisoning by hydrocyanic acid, 187.
  of mustard gas poisoning, 138.
  of poisoning with ammonia, 281.
  produced by lewisite, 159.
  produced by toxic smokes, 176.
System in bathing, 228.

Tachycardia in gassed cases, 247.
Tactical use of chemical agents, 68.
  use of lachrymators, 168.
  uses of chemical warfare, 233.
Tanks, as gas proof shelters, 223.
  possible use of, in transporting gassed cases, 240.
Tear gases, 167–172.
Temperature, absolute, 16.
Test for sensitivity to mustard, 150.
  to determine fitness for duty, 119.
Testimony of Dr. Francine before Senate Committee, 255.
Testing for gas, method of, 209.
Tests, chemical for phosgene, 80.
Thermite, 194.
Thermometer shelters, 46.
Thunder showers, 58.
Tissot mask, 199, 200.
Tornadoes, 59.
Toxic smokes, symptoms produced by, 176.
Toxicity of carbon monoxide, 189.
  of mustard, theories for, 132.
  relative of pulmonary irritants, 83.
Treatment of cases gassed by irritant smokes, 182.
  of carbon monoxide poisoning, 190.
  of lewisite poisoning, 164.
  of mustard casualties, 153.
Trinitro toluene, 38.
Transportation of cases gassed by vesicants, 235.
  of cases gassed by pulmonary irritants, 234–236.
  of gassed cases, rules for, 241.
Tuberculosis in gassed cases, 248, 251, 252, 254, 256–258.
Turkey red oil, 225.

Unit gas officers, 216.
Urease, use of, in phosgene poisoning, 115.

Vapor pressure, 14.
Vertical temperature, gradient, 44.
Venesection in phosgene poisoning, 99, 105, 113.
  versus oxygen in pulmonary oedema, 111.
Vesicants, 69, 125.
  first aid for, 235.
  naval use of, 267.
Vincennite, 184.
Vindictive, attack by H. M. S., 265.

Water, chemical examination of, for arsenic, 183.
  from shell holes often contaminated, 229.
Water, poisoning of, by irritant smokes, 183.
Weather Bureau, United States, 59.
  effect of, on use of gas, 217.
  predictions, 59.
Wind velocity, estimation of, 47.

Xylyl bromide, 72, 167.

Yeast, action of mustard on, 133.
Yellow cross shell, 73, 125, 174.

Sans Tache

Sans Tache

IN THE "elder days of art" each artist or craftsman enjoyed the privilege of independent creation. He carried through a process of manufacture from beginning to end. The scribe of the days before the printing press was such a craftsman. So was the printer in the days before the machine process. He stood or fell, as a craftsman, by the merit or demerit of his finished product.

Modern machine production has added much to the worker's productivity and to his material welfare; but it has deprived him of the old creative distinctiveness. His work is merged in the work of the team, and lost sight of as something representing him and his personality.

Many hands and minds contribute to the manufacture of a book, in this day of specialization. There are seven distinct major processes in the making of a book: The type must first be set; by the monotype method, there are two processes, the "keyboarding" of the MS and the casting of the type from the perforated paper rolls thus produced. Formulas and other intricate work must be hand-set; then the whole brought together ("composed") in its true order, made into pages and forms. The results must be checked by proof reading at each stage. Then comes the "make-ready" and press-run and finally the binding into volumes.

All of these processes, except that of binding into cloth or leather covers, are carried on under our roof.

The motto of the Williams & Wilkins Company is *Sans Tache.* Our ideal is to publish books *"without blemish"*—worthy books, worthily printed, with worthy typography—books to which we shall be proud to attach our imprint, made by craftsmen who are willing to accept open responsibility for their work, and who are entitled to credit for creditable performance.

The printing craftsman of today is quite as much a craftsman as his predecessor. There is quite as much discrimination between poor work and good. We are of the opinion that the individuality of the worker should not be wholly lost. The members of our staff who have contributed their skill of hand and brain to this volume are:

Composing: Andrew Rassa, Ray Kauffman, John Dotterweich, Benjamin Armiger, Roland Stultz, William Kidner, William Fite, William Koch, George Behr, Harry LaMotte, Herbert Leitch, Zeddie Breithaupt, Kurt Woernle, John Flannigan, Joseph Weimer, Harry Harmeyer, Henry Shea, Walter Phillips, Richard King, Edward Rice.

Keyboard: Anna Kelly, Catherine Kocent, Hannah Scott, Harry Susemihl, Vera Taylor, Eleanor Luecke, Minnie Foard.

Press: Leo Ledlich, Henry Augsburg, George Lyons, Thomas Schreck, Clarence Ridgeway, Robert Gallagher, Fred Lucker, William Harrison, Jr.

Folder: Lawrence Krug.

Caster: Kenneth Brown, Ernest Wann, Mahlon Robinson, Charles Aher, George Smith, Theodore Nilson, Frank Malanosky, Martin Griffen, Henry Lee.

Proof Room: Arthur Baker, Alice Reuter, Mary Reed, Sarah Katzin, Ruth Trieschman, Lucille Bull, Ethel Strasinger, Angeline Eifert, Audrey Tanner, Edna Clark, Lewis Forney, Dorothy Strasinger, Lillian Gilland.

Some Recent Books

Ability to Sell

What sales-manager wouldn't like to spot it? What enterprising individual who hasn't asked himself whether or not he has it? In these days of competition, when salesmanship means so much to business, the *ability to sell* is an important qualification. Dr. Merrill Jay Ream's little book under this title tells a big story—worthy of careful study. Based on certain experimental tests in actual selling.

Cloth, $1.25 postpaid Published 1924

Job Analysis and the Curriculum

Are you training young men for executive and supervisory positions? Are you aiming at development into such a position? More and more, the industrial world senses the crucial importance of job analysis. This book by Edward K. Strong and Richard S. Uhrbrock has special reference to the training of printing executives. A bibliography of job analysis is subjoined.

Cloth, $2.00 Paper, $1.00 postpaid Published 1923

Popular Research Narratives

A group of fifty-five minute stories of research invention or discovery, directly from the "men who did it," pithily told in language for laymen, young and old. They were collected by the Engineering Foundation, of New York City. Give the busy man a better idea of what's doing in the scientific world than hours spent in diligent digging. Admirable adventure stories for the youth in school or industry. A special arrangement with Engineering Foundation makes possible a

Price of 50c. 160 pages, cloth bound

Common Sense of Theory of Relativity

Yes, it has common sense to it. Dr. Paul Heyl's little book brings it out, and by apt and vivid illustration enables any mind of intelligence to get the fundamental idea Einstein presents. A little book which you can read in half an hour; but one which you will want to read many times.

Cloth, $1.00 postpaid

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on or before the date last stamped below.

MAR 15 1963
JAN 1 1982
AUG 26 '27
NOV 14 '27
JAN 24 28
MAY 13 28
OCT -2 28
OCT 29 1931
JUN -6 1933
MAR 10 1937
JAN -3 1942
APR 14 1942

LANE MEDICAL LIBRARY OF
STANFORD UNIVERSITY
300 PASTEUR
PALO ALTO, CALIFORNIA

I1246 Vedder, E.B.
V41 The medical aspects of
1925 chemical warfare

NAME 64816 DATE DUE

M. A. Seidenfeld
Seidenfeld
Dr. Eloesser
Seidenfeld

I 1246
V41
1925